THE SURGICAL CLINICS OF JOHN B. MURPHY, M.D.

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THE SURGICAL CLINICS OF JOHN B. MURPHY, M.D.

AT

MERCY HOSPITAL, CHICAGO

OCTOBER, 1912

UNIV. COLL. HOSP. MEDICAL SCHOOL SHL WITHDRAWN LIBRARY

PHILADELPHIA AND LONDON

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REMARKS ON ANESTHESIA MADE AT CLINIC

DR. MURPHY: The sister has occupied the position of anesthetist in this hospital for thirteen years. She has given anesthetics to about 15,000 to 16,000 patients, and she has not had a single fatality that could be attributed directly to the anesthetic. We have occasionally had pneumonia develop following the administration of the anesthetic, but these cases have been very rare. We believe that this excellent record is due to the extraordinary care taken by one individual who is held responsible for that work. She is, in the first place, very well informed on the subject. She reads practically all the literature that is written on anesthetics and anesthesia. She has been trained for this position. It is her life-work.

The sister sees the patient before the operation. She looks over the history to determine whether or not the report on the urine is correct. She asks the patient about the condition of the nose and throat; whether the patient has had morphin or atropin injections or just atropin. Where we want great relaxation we give morphin ($\frac{1}{6}$ grain) with the atropin ($\frac{1}{100}$ grain). Where we want merely suppression of the secretion in the upper air-passages and bronchi we give the atropin only. We are firm believers in the advantages of the atropin. We believe that it very materially lessens secretion. We believe that if we can lessen the quantity of secretion from the nose, throat, trachea, and bronchi, it materially aids in relieving any irritation that might occur lower down during the administration of the anesthetic. We have no confidence at all in the statement that if you give any of these drugs you lessen the expectorating ability of the patient on the table. A patient has no expectorating ability on the table. The idea is to keep the fluid out, and then it does not have to be expectorated. We do that as a routine, and we believe that since we have followed that plan we have had better results.

Furthermore, the sister sees practically all the cases before I get out of the hospital at noon, and if a patient is not in good shape, I get a personal report from her of that patient's condition. The next morning, or in the night, if necessary, if anything has occurred, the sister notifies the intern or me. She becomes the watch-dog of patients and she is there every day.

Contrast this plan of giving anesthetics with the methods that have been followed in other large hospitals for one-quarter of a century or more. In the first place, we find that formerly anesthetics were given to patients by men with the least experience—perhaps the youngest intern. In the second place, we have seen ether poured on by the pint. That is to say, he smothers the patient with ether. He gives five or six, or perhaps twenty, times the quantity that is necessary; he has no idea when the patient is really asleep.

I speak of these things because I was in the same position once myself, and I know that I had the greatest confidence in my ability to give anesthetics, and that confidence was increased and strongly grounded in me by my inexperience and my absolute lack of knowledge of the situation. It made me a dangerous element. I remember giving an anesthetic to a patient who was being operated on by Dr. Gunn. He said to me, "Doctor, that patient is in bad shape." I replied, "I do not think so." It took two hours to get that patient awake! He was cyanotic and nearly dead, and yet I did not have the slightest sense of danger in this case. But Dr. Gunn was kind to me. I learned my lesson that day, and the longer that patient was kept without waking, the more anxious I became. Dr. Gunn did not say very much, but he "looked a lot," and his look was enough for me. It meant something.

In managing hospitals it is also of great economic value to have a trained anesthetist. In the first place, in this institution we save more ether than it would cost to support two or three sisters. In the second place, the patients never have sore tongues following anesthesia, because the sister never permits anybody to put an instrument on the tongue. To give you an illustration: One of the visiting doctors, who was formerly an

assistant of mine, was given charge of administering the anesthetic toward the end of an operation while the sister started to anesthetize the next patient. He had a forceps on the tongue of the patient. It shocked her very much to see that instrument placed on the tongue. If any of you have ever been unfortunate enough to have had a heavy hemostat put on your tongue and kept on there for twenty or forty minutes, and then experienced the discomfort for the next three or four or ten days, you know what these words mean.

In the next place, you should not only consider the great economic value of this method or system, but there is less risk to the patient, which should take precedence over everything else. We must keep that risk to the patient the dominant beacon light of our every act. The risk to the patient, or the lesser risk of pneumonia, is enormously reduced by having a special or trained anesthetist.

The variety of anesthetics you may use is very great. You may use a local anesthetic, which is less risky probably than any other. You may use nitrous oxid, which has very little risk, and which can be given in operations that last one-half hour or three-quarters of an hour, or even an hour, without accident. You may use ether alone, or ether combined with the nitrous oxid, or ether, nitrous oxid, and oxygen.

I was going to say that you may use chloroform, but I think I would put it another way: You should not use chloroform. I am not speaking as one without chloroform experience. I had ten years' service at the Alexian Brothers Hospital in this city, one of the large hospitals here, where we used nothing but chloroform, and I never saw a death from that anesthetic. However, I want to say this, that I kept up artificial respiration once for three hours and fifteen minutes, the longest three hours and fifteen minutes of my life. But the patient lived.

I remember very well one morning going into the Alexian Brothers Hospital and meeting my associate, the late Dr. Henrotin, who was coming out, looking white and pale, in place of having the thoughtful look and smile which he usually had. He looked very much perturbed. I said to him, "What is

the matter, doctor?" He replied, "I just lost the only child, a boy of eight years of age, of one of my dearest friends, from chloroform." Does any one know what that means—what a direful calamity that was? How did he die? He died before he was asleep. That is just it. These patients die before they are asleep.

This recalls the unpleasant experience I had with chloroform, where I kept up artificial respiration for three hours and fifteen minutes. I went over to let water flow on my hands, turned around, and saw a change in the patient's face. In thirty seconds she was apparently dead. Her heart had stopped beating, her respiration had stopped, and she was to all intents and purposes dead. We started artificial respiration, then we resorted to dilatation of the rectum. When the rectum ceased to respond to rhythmic dilatation for six or eight minutes, her respiratory center being out of commission, we went to the upper sphincter of the sigmoid, put our thumbs away up, and ten times in a minute we would dilate to see if she would make an effort at inspiration. The heart kept on doing its work, the pallor diminished, but the minute we stopped dilating respiration ceased. We changed to the vagina, dilated that ten times in a minute, and we kept that up until she finally started in voluntary respiration. Her pulse was 200 the next day, but with care and watching closely she got well.

The danger of chloroform lies in the fact that we do not know definitely what happens. We do know that something does happen, but we do not know definitely yet what patient it is going to happen in, and when practitioners say that in this renal case or that renal case, or in this or that condition, "I use chloroform," I feel like asking the question, what do you know about that patient's ability to stand chloroform? Absolutely nothing. Ether given with oxygen, ether given with nitrous oxid, ether given alone, if administered with caution, is not any more irritating to the kidneys, we believe, than is any other anesthetic. In our experience of ten years at the Alexian Brothers Hospital we watched the kidneys of our patients very carefully,—just as carefully as we watch them here,—and we be-

lieve that the effect on the kidneys is about the same from chloroform as from ether.

In our work it is our endeavor to do the operation in the shortest possible time consistent with good work, thus making the anesthesia as short as possible. We have not used local anesthesia as we would like to do, because we have had such perfect satisfaction with ether as a general anesthetic. We believe that hernias in the aged should be operated with a local anesthetic. So that in the matter of anesthesia in this hospital, we feel fairly comfortable. We feel that the matter of anesthesia here is settled.

To the sister's mind the giving of anesthetics is to *live* in the *doing* of the work. She says: There is no set form, no code of laws, that one may adopt, for the individuality of every case is so marked that no two patients can be treated exactly alike. The service of the anesthetist must necessarily be a personal one. The physician and the scientific value of the work suffer most if she is not mistress of her art.

A few points may be mentioned merely to emphasize the fact that it is not the mere rendering unconscious of a patient that defines anesthesia, but the results that justify methods and the experience-taught lessons. Anybody can put a patient to sleep, but it takes a skilled anesthetist to know how long to keep him asleep, and what his condition under anesthesia is. Never, for one moment, should the patient be lost sight of.

The mouth and nose should be thoroughly cleansed. A small dose of morphin and atropin is given, in order to forestall any accumulation of mucus and to prevent post-operative coughing. These drugs should not be given indiscriminately, as they have a nauseating effect on some people. Good judgment must be exercised in each case, and the benefit of the patient considered first and foremost.

The anesthetist should approach her work with the composure and interest necessary to render the best possible service to her patient, who must be made to feel that she is his friend—one who is determined to do her utmost to assist him and to help him through one of his great ordeals, as patients are wont to

consider operations, even though they may be slight ones. The principal remuneration of the work lies in the knowledge of the fact that the patient is in good condition after the "anesthetic stage," and that the "coming out of ether" is accompanied by as little nausea as can be expected. In all laparotomy, goiter, and brain cases *oxygen* should be given during the general anesthetic. Gas is certainly most advantageous, if a suitable simple apparatus can be obtained, by which a steady, uniform stream is assured. The patient should be kept in as normal condition as possible after operation, moderately warm. A surplus amount of heat is detrimental, as is too much cold.

Chiefly essential among the aids to establish the success of a well-given anesthetic is to labor for the confidence of the patient. If he has implicit, child-like faith in the ability of the anesthetist and her intention to be kind and watchful, it will relieve his mind of much useless worry, the absence of which is an absolute and undeniable necessity, as all will appreciate who are familiar with the details of this tremendous undertaking.

The anesthetic is begun with gas. When the patient begins to lose consciousness, the change is made from gas to ether, which is given with an Esmarch mask by the drop method. When anesthesia is complete, just sufficient ether is given to maintain sleep.

This subject is so extensive that it is impossible to give a fair outline. The work is better done than described with any true degree of accuracy, mainly because of the variety of cases and the difference in the handling of each particular case.

NEPHROLITHIASIS

HISTORY

Miss S., aged twenty-three, housemaid, came to hospital because of recurrent attacks of pain and continuous tenderness in left kidney region.

Family history negative. Had measles and whooping-cough as a child, with good recovery. Has had none of the acute infectious diseases during adult life.

Present Trouble.—When six years old she had an attack of colicky pain in the left side, followed immediately by vomiting. She knows nothing about the duration of this attack or other details. Ever since then she has had attacks of severe colicky pain in the left kidney region, radiating to the external genitals. The pains were severe enough to double the patient up, and during the past six years she has had to have morphin at each attack to control the pain. The attacks come on suddenly, last two or three hours, and disappear as suddenly. Almost immediately after the onset of the pain the patient becomes nauseated and vomits, but she states she never has had a chill or elevation of temperature, but cold perspiration comes with every attack. She has never noticed blood in her urine, but urine was darker after each attack, and several physicians who saw her during or following an attack found blood and pus in her urine. She has had no frequency of urination, but has burning at times. This burning, however, is independent of the attacks. On deep palpation the left kidney area is tender and has been tender as long as she can remember. Since the onset of this trouble the attacks have varied in frequency and severity from eight months to two days apart. Lately the attacks have been more frequent and more severe. No gastric symptoms except during

the attacks. Bowels have been regular. No cough or night-sweats. No heart trouble. No loss in weight.

COMMENTS AND OPERATION

This is a classic picture. The diagram shows a stone. It should be a stone from the classic history. The skiagram must always be interpreted with an interrogation, so far as a stone is concerned.

I read a very good article in a British periodical recently which stated that in 8 per cent. of the cases where the skiagram showed positively a stone, no stone was demonstrable at the operation, and that in 6 per cent. of the cases in which there was no stone shown by the *x*-ray stones were found at the operation. So you must take the skiagram merely as a help—an adjunct—in making the diagnosis.

The diagnosis in this particular case is, of course, easy—nephrolithiasis. There has been a long period of disease, a surprisingly long period, for this girl has had renal colic since she was a child of six. When a child she was ill with these recurrent attacks of pain, nausea and vomiting, suppression of urine immediately, increase in urination afterward, the presence of pus continuously, and the presence of blood continuously now.

In addition to that, the final test that can be made at the bedside and without the aid of the microscope is first percussion of the kidney during the attack. It is one of the most positive signs and is always available. When the patient is suffering from an acute obstruction from any cause to the outlet of the ureter, or when the patient is suffering from an infarct in the kidney which increases the tension on its capsule, we can demonstrate that the lesion is in the kidney and not in the appendix, and not in the gall-bladder, by fist percussion over the spine. The value of fist percussion depends on the tension under which the contents of the renal pelvis are held during the attack. Have the patient sit on the side of the bed, or on a chair, and stoop slightly or lean over forward. Place the left hand first over the supposedly normal kidney, and bring the fist down

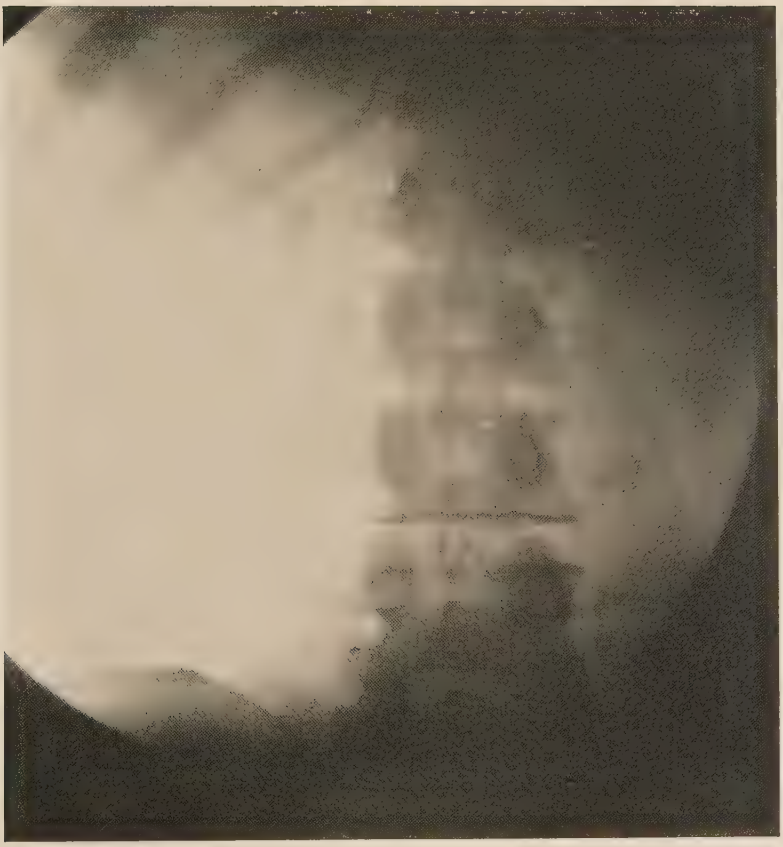

Fig. 123.—Radiogram showing stone in pelvis of kidney. Shadow about one-half inch to the right of the spinal column.

on it, striking a strong blow. There is no response; no manifestation of pain. Change the hand to the diseased side, press it firmly against the site of the kidney, and make the fist percussion, and the patient springs up suddenly and sometimes cries out. There is an immediate manifestation of pain. It is one of the most pronounced responses that I know of to physical tests of diseases inside the wall of the abdomen.

I recall the case of a lawyer on whom I made that test. He came from Gary, Indiana; he was an Irishman, and when I struck him he wheeled around and said, "Doctor, I could hardly keep from striking you—you know that hurts." I merely cite that to illustrate to you how responsive that test is. [These diagnostic methods are described and illustrated in June number.]

In this case for operation today we were unable to palpate the kidney. That means we will probably have some difficulty in bringing it down. We had the mate to this kidney the other day. The contrast in the train of symptoms is marked. That patient had recurrent attacks of pain; had diminution of the quantity of urine during the attacks, with an increase in the quantity of urine after the attack. There was pus in the urine, but she never had the typical colic described in this case. During the attack she had a typical Dietl's crisis. In that case there was an obstruction due to a valve in the ureter.

We will expose the kidney by means of the flank incision, as we did the other day. It will extend from the middle of the twelfth rib, in front of the erector spinæ, downward and forward. The length of these incisions depends entirely on the requirements of the individual case. The kidney region is quickly exposed.

I am into the fatty capsule. I will palpate for the kidney. It appears to be high up. I have it. I am freeing it from its bed and bringing it right down into the field. It does not appear to be materially enlarged. With a clinical history of a stone in the kidney for something like seventeen years one is surprised to find that the kidney is so little enlarged, particularly when we take into account the fact that the patient has had repeated

attacks of colic again and again. With the upper pole free, the kidney comes readily out into the field.

The next step is to palpate for the stone, to determine whether or not she has a stone, and if she has a stone, where it is. The skiagram shows that she has a stone, and the clinical history shows she should have a stone. The skiagram shows that the stone should be well toward the pelvis of the kidney, but on careful palpation I am unable to feel a stone in the kidney. The fact that she has had repeated attacks of pain would lead one to believe that the stone should be down in the pelvis near the ureter. I have thrown the kidney forward so that I can free the renal pelvis from its fat. There is no infiltration around the kidney. There is a mass near the inner side of the pelvis that feels more like an enlarged gland than it does like a stone.

We have a rule in our surgical work on the kidney always to open the pelvis and expose the ureter and pelvis, and not endeavor to palpate through the kidney from the cortical side, but make our palpation from the other side. At the pelvis we find the first evidence of any infection. There is infiltrated tissue here. That shows that there is an infection in this zone. On opening the pelvis of the kidney I can see at once that it is dilated, and there is also infiltration of the wall of the pelvis. That is a guide to the infection that has been present here for so long a period of time. There is the stone! You remember how much that felt like a gland. It looks like a gland, but it is a stone.

Dr. Fenger advocated opening through the cortex, but this is much better. The dilated pelvis is large enough to admit my finger for a considerable distance. I will open this pelvis until I determine the position of the ureter, if I can. I have it. The ureter is patent. I will pass a catheter down into the bladder and see if we have another stone located in the ureter. The position in which this stone was located justifies exactly the clinical history.

I do not know if this catheter is in the bladder, but I will endeavor to determine. By milking the catheter or "stripping" it, if it is in the bladder, you can draw urine out of it, if the bladder contains urine. The catheter collapses after a drop or

two of urine is milked out, therefore the catheter is in the bladder, but the bladder is empty.

The next step is to palpate that renal pelvis, and always do that from the pelvic side. Dr. Fenger had a great fear of opening the pelvis of the kidney, and his fear was based on his early experience. I cannot palpate any other stone.

I will close the opening in the pelvis with fine catgut. There is no occasion for doing a plastic operation, as I did on the case the other day, where we had a condition of the kidney due to a valve-like formation at the attachment of the ureter to the pelvis, also described by Fenger, but accentuated more particularly by Israel, who wrote the master work on surgery of the kidney. No other work, to my mind, has been equal to that in this particular and in its instruction, because it merely cites the cases, arranged in series, and giving the pathology. He had about 221 cases, as I recollect it now, as it is years since I read the book. The case reports in this book represent 221 accurate observations of the clinical course of the diseases of the kidney, not written in the library, and not written with the pipe. It was a record of facts, and that is what one really wants and what is going to count in the future in medical text-books, for both the practitioner and the student.

I am accurately closing this incision in the pelvis, so that there will be no primary leakage. Fenger, in one of his early cases, had a permanent fistula for which he had to remove the kidney, and that annoyed him so much that you could not get Dr. Fenger to cut into the pelvis of the kidney unless the stone and lesion were readily demonstrable. In our early work on this condition we began opening the kidney from the pelvis, and have followed that plan ever since. When I was working in St. Joseph's Hospital, nineteen years ago, I had my first important case of opening through the pelvis of the kidney and removing a stone.

There will be no drainage necessary here. If this was a large dilatation of the calices; if it were three or four times as large as now, I would put in a drain. We put in just a strand of nosophen gauze outside that line of suture and no other drain.

Let the records show that one strand of nosophen gauze is

placed around the pelvis of the kidney. This nosophen gauze merely acts as a siphon drainage primarily; if urine will come out at all, it will come out along the gauze, but it does not act as a siphon for more than a short period of time. The kidney is dropped back and finds its normal position readily.

The next step is the approximation of the muscles; beginning with the location of the iliohypogastric nerve, we look for it on the inner side of the muscle, as it is here. We pick up the muscle without including it. If we include the iliohypogastric nerve in that suture, the patient will have great pain, and that accounts for so much of the pain these patients suffer following the operation, because the suture includes the nerve.

I am approximating the muscles in the regular way, otherwise I would have a hernia. Every man who has had experience in this line of work knows that hernias do occur following this operation, and they are of a very annoying type. The kidney can come out between the muscles in this position, and have recurrent luxation of it, causing great pain and great discomfort. So we approximate these muscles as accurately as we would approximate the muscles on the anterior surface of the abdomen.

In opening the pelvis of the kidney, if it is a greatly expanded pelvis,—if it is two or three times larger than we found it today,—I would make a resection of the pelvis. You will remember the case of last Saturday, in which the dilatation of the pelvis was as large as a baseball, with the long history of a Dietl's crisis. That patient had a mass in her abdomen here larger than a child's head. In that case we followed the line that we had followed in all our previous cases of this class, now amounting to 14. We did a pyeloplasty. We have made 14 complete resections of the pelvis of the kidney with detachment of the ureter and re-attachment of the ureter, with 14 recoveries and 14 good results, showing what can be done in a plastic way on the kidney pelvis.

In that case I carried the catheter down into the bladder, brought it up through the cortex of the kidney and out onto the surface. In addition to the dilatation of the pelvis, there was a dilatation of the lower third of the kidney. The secreting

portion of the kidney tissue might be of the normal content, notwithstanding the great dilatation. We know that kidneys of this kind should never be removed. Why? Because you are removing practically normal kidney, and suddenly throw all the work onto the other kidney, while if the secreting tissue is absorbed gradually, or destroyed, the other kidney has gradually taken up the work and is fit to go on excreting without additional congestion and additional labor, and the patient is not affected at all by the removal.

VISITING SURGEON: Did you pass the ureteral catheter before the operation?

DR. MURPHY: No. We inspected the bladder cystoscopically, and found urine pouring out from the normal side and pus from the affected side, so that we did not need to touch the ureter. If we did not have that picture, then we would have passed a catheter. These healthy ureters should not be catheterized. I am fearful of the infections that occur from transmission of the catheters in the presence of pus.

CHOLECYSTITIS

HISTORY

MALE, aged fifty-two, married, and has three healthy children. Came to hospital because of repeated attacks of epigastric pain. Family history negative. Denies any venereal disease. Has had none of the acute infectious diseases, but has had sick headaches frequently.

Present Trouble.—Began in November, 1911, with colicky pain in the epigastrium. At the same time he states he had a sense of fulness or distention of the stomach. The pain did not radiate to the right shoulder or back, but was localized two inches above the navel, slightly to the right of the median line. Nausea came on immediately after the onset of colic, and was followed by vomiting. Patient had no chill, no fever, and was never jaundiced. The acute pain subsided in seven hours, and there was an area of tenderness over the gall-bladder for two days. Since November, 1911, he has had 9 such attacks with increasing frequency, the last two coming on within a week.

At 10 o'clock on the night of May 2d the present attack commenced, with severe pain in the epigastrium, followed by nausea. He vomited bile-colored material one hour afterward. Four hours after the onset a doctor was called, who put the patient into a hot bath and gave him some powders. This treatment had some influence on the pain, but very little. He refused a hypodermic. At this or previous attacks he had no fever, no chill, no jaundice, no clay-colored stools, no high-colored urine, no bloody urine. The attacks came on independently of meals, although he states they always followed a hearty meal.

COMMENTS AND OPERATION

DR. MURPHY: Did the patient have increase in frequency of urination or blood in the urine after the attack?

INTERN: No, doctor.

DR. MURPHY: How do you know?

INTERN: He said so.

DR. MURPHY: What was his temperature?

INTERN: His temperature was 100.6° F. up to 9 o'clock last night, coming down this morning to 96° F.

DR. MURPHY: What brought on the attack?

INTERN: The patient says he ate a hearty meal.

DR. MURPHY: Did you say that in the history? I didn't hear that. What about this attack? Let us see. This man came to my office one day this week. I examined him, but from the history was unable to arrive at a diagnosis. There were some statements that were indefinite. Following a meal he had pain, nausea, and vomiting. He never vomited that day. The pain would last six, eight, or ten days, and then disappear. He had very little temperature, very little local sensitiveness, and I could not make a positive diagnosis as to whether the trouble was in the kidney, in the appendix, or in the cholecystic tract. I told him to have a good dinner. He did, and at half-past ten that night he had an attack. It was the most severe attack he has ever had. Then he wanted to take some anodyne for it, and I told him to go through the night, that I would see him in the morning. In the morning he called his doctor, who would not give him an anodyne, and later I saw him with the condition present that I am going to mention now.

He was sensitive under the right costal arch; he could not breathe with my hand pressed beneath the arch, and on perpendicular pressure over the gall-bladder he had severe pain. He was not sensitive in any other position with the fist percussion. He had no pain; he had no blood in the urine; he had a temperature, with leukocytosis. That is the advantage of seeing a man in the attack. The practitioner who sees a patient in the attack has an advantage over the man who sees him in the

interim. That is why he can avail himself of the positive signs of the presence of acute retention of bile. Remember, acute retention gives a train of symptoms valuable in making an immediate diagnosis. All in the way of physical signs disappear as soon as the acute retention is over. That is why you can tell there is an acute retention, and the principal evidence is to put pressure on any place that is under inflammatory tension. It is just like a felon. Why does a felon pain? Not because the nerves are injured, but because the inflammatory products are held under pressure. All the nerve filaments are under superior tension. It is just the same whether it be the appendix, the gall-bladder, or the pelvis of the kidney.

This man never had a temperature before, and I think he is telling the facts as far as he knows. He says he did not think he had much temperature, yet he had quite a temperature, and not only that, but he had quite a leukocytosis. This is as important as temperature, if not more so in an acute attack. The leukocyte count is corroborative of the clinical course. For many years, in all these acute conditions, we had to make a diagnosis without the leukocyte count. We consider it of very great advantage.

The time when leukocytosis is of so much value in practical work is in relation to the appendix. In a small percentage of cases of appendicitis we have, with only a very slight elevation of temperature, say, half a degree or three-quarters of a degree, or even four-fifths of a degree, a leukocyte count increased to 4000, 5000, or 6000, and in that class of cases it is valuable, because it indicates the presence of infection. The temperature and the leukocyte count are very closely related, but in a few cases, and in that few you need it very much, there is a great spread between the leukocytosis and the temperature. Occasionally the temperature will be 103° F., with a leukocyte count of only 8000 or 9000. Again, you will find less than a degree elevation of temperature and a high leukocyte count—up to 14,000 and 15,000. There are two conditions in which it is valuable to you as an aid.

This man has no history of gastric ulcer. He has no history

of ever having had continued soreness. After he gets over a spell of soreness, two or three days after he gets over an attack of pain, he is apparently perfectly well again until the next spell comes on.

I recall the case of a policeman who, every two months, had to lay off duty on account of attacks of pain in his epigastrium. They would come on like colic, and were so severe that he would have to go home. We examined him, but could not make a diagnosis between the attacks. We could not do so until he got the next attack, when I could clearly outline a tumor in his epigastrium—not to the right side, but central, hard, and sensitive. I made a diagnosis of gastric ulcer, which was confirmed at the operation. This case belongs to the exceptional class, so that you must always bear that in mind in making a diagnosis of gastric ulcer, that you may have periodic pains; that they may subside and still be of the gastric ulcer type.

Remember, this man never has had any temperature, never had jaundice, and that is an important factor in all this class of cases. For a long period of time we considered jaundice as essential to the diagnosis of cholelithiasis. I have repeatedly cited my experience here and elsewhere in regard to cholelithiasis, namely, that in all the cases we have operated on and demonstrated gall-stones, in 86 per cent. of them there had never been jaundice at any stage in the history of the case. In only 14 per cent. of the cases that have come to us with gall-stones present has there been jaundice.

We make the usual incision for exposure of the gall-bladder, just below the costal arch, at the junction of the ninth rib with its cartilage, cutting through the abdominal wall, and then the peritoneum. The gall-bladder readily comes into view because of its large size.

I find here that gangrene of the gall-bladder is commencing to take place. He is in the acute stage of gangrene. There is an old infiltration present. You see the yellowness. It is a beautiful specimen, and shows that we must get at these cases early and operate promptly if we would save life. There is not a single adhesion here to protect the gall-bladder, and it is

ready to break into the peritoneal cavity. When a patient has an acute attack like this man has, he is in great danger of losing his life. There is no use trying to palpate the gall-bladder for stones, as there is too much tension there. You see now the wisdom of having this man stay here in the hospital and trying to see him in an attack. He has some temperature. This gall-bladder is septic. It is brittle and under the greatest tension. I will aspirate the gall-bladder and reduce the tension. You see this looks like clear bile. The pus will commence to show when we get further down. There is thick, infected bile with blood in it. Now we have the tension off.

He never had jaundice in all these attacks. Jaundice is a symptom after the stone gets into the common duct, but it is never a symptom before, and a man may have 1000 attacks before the stone gets into the common duct.

We will determine whether there is a stone in the cystic duct,— whether there is a stone present at all,— with the type of colic he has had. One comparatively small stone I have pressed back from the cystic duct. Shreds are forming on the outside of the gall-bladder from the infection. I must examine his common duct and his pancreas. Between my middle and index-finger I have the hepatic artery. I can feel it pulsating. I know that right along that, on the under side, is the common duct, so that I carry that mass with me and know that I am going over the common-duct zone. I now palpate the head of the pancreas.

Let the record show that there is no infiltration of the head of the pancreas; that the glands at the neck of the gall-bladder are enlarged; that there is no stone in the common duct; the only stone that is demonstrable has been dislodged from the cystic duct.

Now we are ready to do a cholecystostomy and drain. We will do it in the regular way. We will not take out this gall-bladder. It is very friable, and we have to handle it with the greatest care, otherwise it will burst and that is dangerous, because it is infected. It is a little surprising to me that the intestine or omentum did not commence to cover over that necrotic zone. That man's life was unprotected. It is a common thing to have the intestine

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or omentum adhere around the gall-bladder, encapsulate it, and care for it, preventing the escape of infective material from the gall-bladder into the free peritoneal cavity. This illustrates a condition that does not occur very often—I will say, in three or four cases in every 100 of the acute ones, or about 3 or 4 per cent. However, they do occur.

I recall the first case of gangrene of the gall-bladder I ever saw. The patient was attacked with severe pain, followed by nausea and vomiting, elevation of temperature, and when I saw him, on the second day of the attack, his temperature was 103° F. He was very sensitive in the gall-bladder zone. We made a diagnosis of acute infection of the gall-bladder, and operated on him immediately. That was away back in the early nineties. The patient had at that time complete gangrene of the gall-bladder clear down to the cervix, but it was not separated and the peritoneum was not dead. We put in a drain. There was only one stone, and that was impacted in the cystic duct. The patient had a virulent infection.

We will now open the gall-bladder. First we insert a purse-string suture of catgut in the dome of the gall-bladder, about one-half inch from the top of the fundus, leaving the ends long. Then we insert a second similar suture, one-half inch lower down, also leaving the ends long. We surround the gall-bladder and pack this entire area with gauze, so that the infected contents will not pass into the peritoneal cavity. I will show you that it is gangrenous. You will see that the mucosa is gangrenous; that it is not the color of the gall-bladder that shows, but it is the gangrenous mucosa. There are a number of stones present, although I felt only one. There are four stones. What a wicked thing that was in such a short period of time.

I am making this opening a little larger for my rubber drainage-tube. Gangrene of the gall-bladder is present already, and it shows how quickly this gangrene takes place, but this is exactly in consonance with our knowledge of gangrene that occurs in connection with appendicitis.

We will turn the mucosa in. We will make an inversion of that cut edge of the gall-bladder, so that any bleeding that may

take place will do so into the gall-bladder. I will fix the gall-bladder at this point to the tube, so that the tube and gall-bladder cannot separate, and before I draw the second row of stitches taut, I will push the tube down into the gall-bladder, inverting or burying the cut edge in the gall-bladder, making an entropium.

VISITING PHYSICIAN: Is that silk you are using, Dr. Murphy?

DR. MURPHY: No, it is catgut.

We will put a second tube into the peritoneal cavity, outside of the gall-bladder. I will stitch that tube to the gall-bladder tube. The needle, in passing through this rubber tube, does not make a hole that will leak; the rubber tube contracts onto the needle, so that there will be no leakage from the needle-hole. This is an extracholecystic drain in the fossa, because that fluid is septic.

We will now commence to make the usual ectropium of the edge of the peritoneum, using catgut. That prevents the formation of adhesions within the abdomen.

VISITING PHYSICIAN: Do you sew the gall-bladder to the parietal peritoneum?

DR. MURPHY: No, not at all. When we started to do gall-bladder surgery there had been only 13 cholecystectomies performed, I believe, up to that time. Then we fixed the gall-bladder to the parietal peritoneum. We were afraid to drop it back as it is dropped back here. Then, as gall-bladder surgery evolved, we fixed the gall-bladder to the peritoneum low down. Originally we fixed it to the skin and had a cholecystic sinus form. As the evolution of this work went on we came to the plan that I think was first introduced by Mayo, that is, of putting a tube in the gall-bladder, suturing it, making an ectropium of the cut edge, and then putting in a second line of sutures, as we did here, and drop the gall-bladder back into the peritoneal cavity. Remember, the tube has only two holes in it—one at the end and the other at the side, near the end. The cut edge of the gall-bladder is turned in and bound around it firmly. The gall-bladder is then dropped down into the peritoneal cavity. This tube will drain everything from the gall-bladder. I put in

a second drainage-tube because there is a septic condition present here. It will drain the duodenohepatic fossa, taking care of any material that escapes from the gall-bladder.

VISITING PHYSICIAN: How long do you leave the tube in?

DR. MURPHY: It will be left in twelve or fifteen days in this case, because a portion of the mucosa will slough and come out through the hole made by the tube. We do not disturb it at all for twelve or fifteen days, and then take it out and inspect the gall-bladder. At the end of five or six hours nature will have encapsulated the tube and shut it off from the peritoneal cavity. At the end of fifteen days you have a connective-tissue canal formed down to the gall-bladder, where the mucosa is showing up on the inner side. There was free fluid in the peritoneal cavity in this case.

Let the record show that the gall-bladder was greatly enlarged; that it protruded well below the costal arch; that it was gangrenous over an area as large as a nickel; that the neighborhood of that was congested, and looked more like liver tissue than like gall-bladder surface; that there were no adhesions whatever to the gall-bladder at the position where it was going to perforate, and if it had perforated, it would have done so directly into the free peritoneal cavity.

On palpation of the gall-bladder a small stone was found down in the cystic duct; this was displaced out of the cystic duct. Three other stones were removed. The patient never passed a stone through the common duct; had never had a jaundice at any time in his attacks. When these patients pass the original stone through the common duct, jaundice uniformly occurs, and sometimes even when the stones are small, because it produces trauma and edema of the mucosa around the stone, and thus a comparatively small stone occludes the common duct and causes a typical jaundice. If the stone has been in a considerable time, the jaundice may entirely subside, and one often finds a stone in the common duct where the patient has not had jaundice for years, but one rarely ever finds a stone in the common duct where a patient has not had a history of icterus at some time in the course of the attacks.

Continue with the record. Further examination and palpation of the common duct revealed no stone. There was no infiltration of the head of the pancreas. The condition of the gall-bladder shows that there was an acute gangrenous infection of an old infiltrated gall-bladder, because the surface of the gall-bladder could not change to the condition it was in in one single attack of short duration, namely, night before last. This patient is getting his reward, because he did what he was asked to do, which accounts for the fact that he will not have a septic gall-bladder rupture into the free peritoneal cavity at 8 or 10 o'clock to-night. It takes the gangrenous portion of the gall-bladder a little longer time to separate than in the case of the appendix. The gangrenous portion of the appendix can separate as early as thirty hours. The gangrenous gall-bladder does not separate as early as that because it has more fibrous tissue in it, and it has not so rich a circulation.

The stomach was free from adhesions, as was also the duodenum. There was considerable free fluid in the peritoneal cavity. It was not very cloudy. With a gangrene of the appendix as severe as that and approximating the surface as near as that did there would be a milky fluid or what we call a purulent fluid in the peritoneal cavity. That purulent fluid, before perforation, is not pus. It is a defensive fluid, and not an offensive fluid, namely, it is lymph filled with polymorphonuclear leukocytes to take up the microörganisms when they come in.

The abdominal wall will be closed with a figure-of-8 stitch. Horsehair will be used to approximate the cut skin edges.

As to the subsequent course of this case: Will we have superficial infection here from the pus and bile that came from the gall-bladder? We do not know. What is the rule? The rule is that we do not. The rule is, with a more purulent material escaping than we have now primary union of the abdominal wall occurs down to the tube, the bile and pus both passing into the wound. Why that occurs we do not know.

The same thing occurs where you have an offensive, rotten material escaping from the surface during the opening of a peri-appendiceal abscess. The infection more frequently pro-

duces necrosis of the tissue in the neighborhood of the tube, but not infrequently, with the most foul material, you get primary union of the abdominal wall where the wound has been smeared with pus. Even with all our care and precautions, occasionally, in preparing the wound and no pus in the field, we find difficulty in keeping it aseptic. So, you see, there are some things about sepsis that have to be cleared up, and, notwithstanding the fact that we sit back and fold our arms and seem satisfied, we have a lot of things to learn why one case does badly and why another does not.

We have learned some things in connection with arteriosclerosis cases. These patients stand infection so very badly that when one has anything like a severe infection in a patient with arteriosclerosis, that patient is likely to die.

We have had in the last few years some striking cases of that in this hospital. Four or five weeks ago a patient came in with hemorrhoids. Following that he had a perihemorrhoidal abscess in the perineum. He was an arteriosclerotic. He began to get an infection, and on Monday, after having got off the train in the morning coming from St. Paul, he went to his home. Later in the day he came for treatment, and in the afternoon, after 3 or 4 o'clock, we saw him. His scrotum at that time was infiltrated and black. His perineum was likewise infiltrated and black, and he had a subcutaneous infiltration extending up to the level of the umbilicus, so that when we cut it open along both sides parallel with Poupart's ligament, we found an emphysematous gangrene extending up to the level of the umbilicus.

Another case similar to that was a preputial abrasion. The man took a walk of a mile and a half on a warm spring morning and got an abrasion of the prepuce. This occurred on Friday. On Sunday evening he had a mild chill; on Monday, a severe chill; and on Tuesday he had another chill. Then he told a doctor about "a little inconvenience" he had. He was treated for malaria, which means nothing at all at that season of the year,—in March, I believe,—because the mosquitos were not alive in the district in which he was living. That statement was undoubtedly made by the doctor to cover an error in diagnosis

or his inability to make a diagnosis. He had a second chill on Tuesday.

I saw him on Friday, and he had complete gangrene of the scrotum, of the corpus spongiosum, and the gangrene extended up, as it did in the other case, to the level of the umbilicus. He had had arteriosclerosis, with angina pectoris, for twelve years, to my knowledge, and had attacks of angina pectoris at night for twelve years preceding, showing the virulence of the infection or the absence of resistance in the arteriosclerosis cases.

When these patients have a mild infection, it is much more serious than a similar infection in younger people, and hence one has to do the work with great caution, and particularly in loose cellular tissue of that variety.

I will ask the intern to reread the history of this case, so that we can make some comments on it. [Here the intern reread the history.]

Dr. Murphy: You can see why we put it cholecystitis in place of cholelithiasis, because he had pain which was not a typical colic. He had the soreness and resistance to percussion and to the "hook" test under his costal arch, and he has leukocytosis and elevation of temperature. This shows you the point we have accentuated so long, that the cases of infection in the gall-bladder have manifestations in the way of elevation of temperature, varying with the location of the infection or the location of the foreign body. When it is in the gall-bladder, you rarely have a temperature which exceeds 101° F. When it is in the cystic duct and there is sepsis present, you frequently have a temperature running to 103°, 105½°, or 106° F. When it is in the common duct, with the stone retained there, and there is infection present, the patient has chills and fever, the same as he has when it is in the cystic duct, with high temperature and icterus added to it. The last is the most dangerous class of cases we have to deal with, because with icterus and sepsis both present, and with a stone impacted in the common duct,—acutely impacted in the common duct,—there is danger of an ascending cholangitis occurring, going clear up into the small bile-ducts, from which the patient can and often does die, with symptoms of what was

called icteric necrosis, with retention of bile and small necrotic foci throughout the liver.

In the other variety, where that does not take an ascending course, intense sepsis is present in addition to the cholemia. That is the class of cases that gave Kehr so colossal a mortality, but that is the class of cases in which you would not operate at this time. If that man had an icterus, with a temperature of 103° to 105° F., all coming on within the last few hours, and you operated on him, he would die, as a rule. I make that statement because it is based on Kehr's statement that he had 97 per cent. of these cases terminate fatally. We do not do operations that have a chance of life of only 3 per cent. It is not good surgery. A larger percentage of these patients will live, get necrosis of the common duct, get pericholelithic abscess, get an abscess in the upper abdomen, and still live, with a subsequent drainage, in more than 3 per cent. of the cases when interfered with in the acute stage.

Here in this case it was different. There was no jaundice and had not been. Here we had all the conditions showing retention of infection under pressure, with only a little absorption. When there is this great pressure in the gall-bladder, why is it you do not have as high a temperature as you do when there is a little stone in the cystic duct, or in the common duct with infection present? Because the fundus of every viscus has but few lymphatics. The fundus of the gall-bladder, the fundus of the urinary bladder, and the fundus of the stomach have but few lymphatics. They do not get the sepsis from there. The absorption occurs only where there is a rich lymphatic supply, beginning at the infundibulum of the cystic duct and extending down to the ampulla of Vater. That rich lymph supply admits of the rapid absorption of septic material, chills and fever, with recurrence, resembling the recurrence of chills and fever of malaria.

When I was a student, acute malignant icteric malaria was regularly diagnosed as a stone in the common duct, with chills and fever that came on every second or third day, like the chills and fever of malaria, and the first man in this country, to my

knowledge, to elucidate that subject was Dr. William E. Quine, of this city. He reported four postmortem examinations in which he demonstrated stone in the common duct, with the typical classic condition reported, and as described in Trousseau's Internal Medicine, as malignant malaria, due to what? Due to the sepsis from a stone impacted in the common duct.

GASTRODUODENAL ULCER—GASTRO-ENTEROSTOMY

HISTORY

MALE, aged forty-one, farmer. Drinks moderately. Smoked to excess up to four months ago; since then has been chewing. Denies lues or gonorrhea. Had pneumonia when a child. Right clavicle fractured twenty years ago.

Present Trouble.—Began five years ago. Once a year, during the first three years, he had attacks of gastric pain, lasting five or six weeks. The fourth year the attack lasted three months, and this last year it lasted seven months. Began May, 1911, and lasted until December, 1911. Pain came on from thirty minutes to four hours after eating, generally in from two to three hours. It lasts until he vomits or until the next meal, when eating relieves it. It is in the epigastrium; does not radiate. He wakes up sometimes at 12 P. M. or 1 A. M. Solid food causes more distress than liquids. Soda gives some relief. Nauseated four hours after eating, and only vomits when he gags himself. Vomitus is sour; contains no blood, but has contained food eaten twelve hours before. Belches a great deal of gas. Has a regurgitation of sour fluid. Not tender over his stomach. Never fainted. Bowels constipated; stools never black, except when taking medicine. No headaches. Has lost 28 pounds in the past eight months. Since December, 1911, he has not had pain, but has heavy feeling in stomach and it is distended from thirty minutes to two hours after eating. This lasts until the next meal, when he is relieved for from thirty minutes to two hours after eating. Nauseated twenty minutes to one hour after eating. Never vomits unless he forces himself to do so. Skin had a yellow tinge two months ago. Heart and lungs negative.

Examination.—No tenderness in gastric area or elsewhere in abdomen. Gall-bladder is absolutely free from tenderness on deep percussion and finger pressure. No rigidity or tenderness of spinal column. No signs of locomotor ataxia. Left iris reacts only slightly, due to trauma to eye fourteen years ago, with adhesions. There is an area of tenderness on deep percussion which can be found with difficulty at a point corresponding to the pylorus. It is probably a saddle ulcer at pylorus.

COMMENTS AND OPERATION

DR. MURPHY: The history extends back over a period of five years, with how many attacks?

THE INTERN: Seven attacks, doctor.

DR. MURPHY: What were the abdominal symptoms?

THE INTERN: Pain two or three hours after eating.

DR. MURPHY: Lasting how long?

THE INTERN: Until the next meal.

DR. MURPHY: Relieved by what?

THE INTERN: By eating and vomiting when he gagged himself.

DR. MURPHY: Relieved by emptying his stomach or eating. What about the night disturbance?

THE INTERN: He would wake up about 12 midnight or 1 A. M., and turning on the left side would give him some relief.

DR. MURPHY: Turning on the left side would give him relief?

THE INTERN: Yes, doctor.

DR. MURPHY: What else? The first attacks came on what time of the year?

THE INTERN: In the spring.

DR. MURPHY: The first attacks came on in the spring and fall. The rule is they always come on either in the spring or fall, rarely ever in midwinter or in midsummer. That fits exactly with the common course of these lesions. Now what else? What other symptom have you not accentuated in your history?

THE INTERN: Feces examination showed blood chemically, and stomach-contents showed blood.

DR. MURPHY: But he never had a severe vomiting of blood;

never had a hematemesis, and he never had a melena; never had a large quantity of blood discharged from his bowel.

THE INTERN: No, doctor.

DR. MURPHY: What other symptom have you not mentioned with force? What other symptom do these patients have? the symptom that is so characteristic of these lesions?

THE INTERN: Localized pain. He had that, doctor.

DR. MURPHY: Remember, the train of symptoms are pain relieved by taking food. If the ulcer is inside the pyloric zone, pain comes on earlier than when the ulcer is outside the pyloric zone. If the ulcer is outside the pylorus, in the first portion of the duodenum, pain occurs two and a half to four hours after eating. When the ulcer is inside the pylorus, you have pain increasing up to the intake of food; when the ulcer is outside of the pylorus, you have pain relieved by the intake of food. He says this patient had to get up at night about 12 or 1 o'clock, which is a common thing in duodenal ulcer, and if he took sodium bicarbonate or a glass of milk, or even a glass of water, he was temporarily relieved. We have all believed that food relief is due to the fact that the food stops coming through the pylorus. We do not know that that is true, and it does not matter.

The next symptom of importance is the appearance of blood in the vomitus and in the stools, either microscopically or macroscopically. With a severe duodenal ulcer the patient may vomit the blood as well as pass it in the stools.

These patients get up in the morning feeling as well as usual and go to work as usual; suddenly they commence to feel discomfort in the abdomen; feel faint, do faint, and then vomit a large quantity of blood, with loose bowels for two and a half to three hours. So classic is that history that it guides one at once to the diagnosis, and that is why the doctor mentions that this patient never fainted and has no history of a severe hemorrhage.

Then we go on to the next symptom—the loss of weight. The loss of weight is usually not great. You will find that these patients with ulcers for weeks and months and years maintain about their regular weight. This man has lost 28 pounds in six

months. We charge up part of that loss to the restricted diet he had been on, but the restricted diet does not begin to take off 28 pounds. There is something else wrong.

Next is the symptom of gas. It is one of the conspicuous symptoms of ulcer, coming one, one and a half, sometimes two hours after the intake of food, and it comes in large quantities; the patient becomes distended. Following the distention the patients will tell you that if they get up large quantities of gas they are relieved. It takes the tension off the ulcer, that is why the patient is relieved.

Pain in the duodenal type of ulcer, as a rule, is never acute. Pain in the gastric type of ulcer can be very acute and resembles a colic, but in the duodenal type it is more a sense of pressure and disturbance in the epigastrium than a real acute pain. One of the other conditions associated with the train of symptoms is pain. Pain after the intake of food, increased by the further intake of food, and not relieved by the intake of food, and continues for two and a half and three hours and then is most pronounced two and a half or three hours after the intake of food, with sensitiveness in the epigastrium, is the pain of gall-stones. You remember the case we operated in our last clinic. The woman's symptoms were associated with food intake coming on two and a half or three hours after meals. She had attacks of pain, not severe enough for colic, but a severe pain and distress after meals. On operation we found an overdistended gall-bladder of very large size, with grumous bile. She had gangrene of the gall-bladder. That was the appalling thing, and with symptoms so mild. The gall-bladder was absolutely gangrenous, without a single adhesion or a thing to prevent it from rupturing into the free peritoneal cavity. That accentuates the relationship between the gastric symptoms and the overdistended gall-bladder, rather than gall-stones.

I will make the usual incision through the rectus muscle. We lift up the rectus sheath on the inner side, and displace all the muscle-fibers upward. Lifting that up in this way we go to the linea alba and simply displace these fibers. I am down on the deep rectus sheath. When I suture that sheath the muscle will

fall back again to the central position. I always cut the peritoneum just below where it is grasped with two tissue forceps, not between the forceps. Always cut on this slanting line. As the doctor pulls up you will see that when the abdomen is opened the omentum and stomach slip out of the field so that they cannot be cut. There is the stomach now in the field. I put on these towels to protect the abdominal contents from coming in contact with the abdominal wall. Then we lift up the bowel. The ulcer is plainly to be seen. It is right at the pylorus. See the white line! There is where the ulcer endeavored to perforate. I will see whether it goes out into the duodenum. No, the ulcer is on the pyloric side. The patient came to us against the advice of the medical man. He came just in time. The ulcer would soon have perforated. There is the result of an ulcer of five years' duration. The spot where it has endeavored to come through is a hard mass.

We must first take care of that perforation and sew over it the omentum. I sew it over the indurated area, so that it will be protected and heal. The omentum will seal all that place and prevent leakage during the early hours after the operative procedure. I am just putting on an omental patch there. Dr. N. Senn is the man who worked out that patch idea originally, and all the patchwork is based on Senn's original experiment. I am going away beyond the line of induration to catch up my patch. This is the best class of case for operation. The ulcer is big, it is near the pylorus, and interferes with the outline of food.

The next step is to do a posterior gastro-enterostomy. There is the ligament of Treitz, and there is the first portion of the jejunum. The ligament that is attached to the first portion of the jejunum is visible.

The next step is to expose the posterior wall of the stomach and open the lesser peritoneal cavity. I will make that opening large enough so as to bring the posterior wall of the stomach into the field, and I want to bring it out as near to the pyloric zone as I can. I will lift the posterior portion of the stomach out of the peritoneal cavity, and that will close the lesser peritoneal

cavity so that the intestine cannot find admission to the lesser peritoneal cavity following the operation and become strangulated. This is the first step—to see that that accident cannot occur, and that that line of union is not going to be hazarded by anything intervening. That portion of the stomach cannot any longer recede. There is the portion of the stomach to which I am going to make my attachment, not transversely, but in a downward direction with the food current.

Now we will empty the jejunum downward and put on the intestinal clamp in the regular way.

I am getting ready for the combined operation of gastro-enterostomy, a different operation from that we have been in the habit of doing. It is the combined operation of button and suture. I will show you how easily that can be done.

The greatest danger in a gastro-enterostomy is hemorrhage. Everything here is protected, however. We use two straight needles threaded with Pagenstecher linen to make the intestinal suture. We use the Hartmann holder for steadying the button, after having put in the stitch. The suturing is done by passing these long straight intestinal needles parallel to each other through the bowel, and then the bowel is incised between the needles. We are then ready to insert the button. Half of an oblong button is inserted into jejunum and stomach respectively.

The next is to take up the stomach. We pull out the stomach and put the clamp on it and get it up as much as we need for the purpose of inserting the button. We insert the needles here just as we did in the case of the intestine; take up all the coats of the bowel in the stitch. It is the same proposition as before—divide the stomach between these two needles. After the two halves of the button are in place, we pull the suture taut so that both stomach and bowel are held firmly against the button. Now we are ready to make the approximation. We press the two halves of the button together tightly, catching stomach and bowel between them, so that there will be no leakage. Then we take off the clamps. The approximation is perfect.

Now we commence to put in the tacking stitches on the side. I will show you how quickly you can run a stitch all around

the line of union and insure against hemorrhage and against leakage. Usually we just put in a single tacking stitch here and there all the way around. In place of that we will put in a continuous stitch around the circumference of the approximation. You can do this operation in any combination you desire.

While in Europe a year or so ago we saw a great many gastro-enterostomies done, but no two men were doing gastro-enterostomies in the same way; at the same time all were getting good results. With the curved needle used by Moynihan, even though it is a large needle, you can get in almost any position in this region that you desire.

What will be the subsequent course of this case? It should be that he will have a drainage here; his food in the future will not pass over the ulcerated zone, but it will be short-circuited. Now we drop that back into the abdomen. It falls back into the normal relationship of his jejunum and stomach, except that in the future there will be no interposing mesocolon between his stomach and his jejunum.

The next thing is to suture the peritoneum by making an ectropium of the serosa. You can see how I make that ectropium of the serosa, by simply passing the needle all of the time inside the edge. That was the combined suture and button operation, with the continuous suture. The abdominal wall is closed in the usual manner—with deep and superficial combined figure-of-8 catgut and horsehair skin sutures. The wound is dusted well with bismuth subiodid powder and sealed with collodion. The usual abdominal dressing is put on. In this case we also sutured separately the posterior and anterior rectus sheaths.

Let us go over the history again, while we are looking backward at our diagnosis, after we have had "the proof of the pudding," and see how it fits with the symptoms, how it fits the symptoms of a gastric ulcer when the ulcer is of long standing.

He had evidence of hyperacidity, which is so characteristic of ulcer in the pyloric zone—more common, I would say, when the ulcer is outside the pylorus than when it is inside. I think it can fairly be translated in this way: that the gastric hyperacidity, as it is commonly called, is caused by ulcer in over 80 per cent.

of the cases. Hyperacidity is to be interpreted differently from what it was before. We are in the next five years going to change the word hyperacidity to ulcer in the neighborhood of the pylorus. The hyperacidity is merely one of the symptoms. So commonly is the ulcer associated with all the conditions known as hyperacidity, and so commonly is hyperacidity made as a diagnosis, while hyperacidity is a symptom and not a condition at all. That is the proper way. There are many conditions that cause hyperacidity, but the most common is ulcer.

This man had his pain two or two and a half hours after his meals. That is exactly right for the location of that ulcer, and exactly the same as if the ulcer were outside the pylorus, in the duodenum. We have endeavored to accentuate the fact that the difference between these two conditions is not sufficient to make a differentiation.

This is a very beautiful illustration of what we find in this class of cases. These ulcers do not heal under medical treatment in the large percentage of cases. In about 70 per cent. of the cases we get permanent healing with gastric drainage of the type I have performed. In about 15 to 20 per cent. we get a great improvement in the condition. In the other 10 or 15 percentage we have recurrence of the ulcer, but that is the exception.

That ulcer was exceptionally large. It was very thick, and that is just the sequence of five years' effort on the part of nature to repair the lesion, and it does there just what it does in so many positions in the body. Wherever you have chronic ulcer you have constant coffer-damming. You saw the anemia of the central zone—the white patch, that is where the perforation occurs.

The button will slough out as healing takes place, and will be passed with the feces. Sometimes it passes soon after the operation, but occasionally it is not passed until late. In a recent case it was passed on the eighty-first day. [In this case the button was passed on the eighteenth day.—Ed.]

APPENDICEAL ABSCESS

HISTORY

This girl is a student, fifteen years old. She entered Mercy Hospital because of pain and excessive tenderness over the entire right iliac region.

Present Trouble.—The present trouble began two weeks ago. She had a headache and sore throat, and insists that she felt feverish, but had no pain in any part of her body. That was on Thursday. The next day she stayed in bed and felt feverish and at times chilly. Saturday she remained in bed, also on Sunday, when she vomited twice. On this day she experienced severe cramp-like pains about the center of the abdomen. These continued until Wednesday, when she had pain for the first time in the right iliac region. Her temperature on this day was 100° F. She has been in bed ever since and has had a temperature continuously. A doctor who saw her late Friday elicited tenderness in the right iliac region. Leukocyte count, 12,000.

COMMENTS AND OPERATION

Dr. Murphy: How long after she had the initial symptoms did she vomit?

The Intern: The trouble began on Thursday.

Dr. Murphy: The trouble began on Thursday? When did the pain begin?

The Intern: She began with symptoms of the grip on Thursday.

Dr. Murphy: I want to bring that point out because of the class of cases which we had in Chicago this last winter. It is a new type of cases to me and to the whole medical profession. The epidemic was most pronounced in Chicago, next in severity in Philadelphia. There have been a number of cases of peritonitis from disease of the appendix—a slight edema and swelling of the

appendix, congestion of the mucosa; an extensive reddening of the peritoneum. It all began with this type of septic angina. The patient had a sore throat and headache and all the evidences of grip. This is what it was called in the early part of the season, but we learned later that it was a peculiar and apparently specific type of infection due to a coccus resembling the diplococcus of pneumonia, but it was not the diplococcus of pneumonia, so far as we were able to learn. I recall two families where the disease affected every member of the family; each one in each family had a typical attack of infection of the peritoneum, as indicated in this case.

In all these years I have talked to you about appendicitis pure and simple. I have told you that appendicitis always began with pain, nausea, and vomiting. Here is a type of case in which the trouble did not start in the appendix. It began as an angina, and then the appendicitis came on with the regular cycle of symptoms.

We lost one of our patients. I operated and found that condition of the appendix—that peritonitis. She recovered from the peritonitis and finally developed a pneumonia. She developed tympany, which is so commonly associated with pneumonia.

We had another case that began in practically the same way. The patient had recurrent attacks of appendicitis and came into the hospital on the third or fourth day of the appendiceal trouble. It was a clean-cut case. The late Dr. Henry Lyman recognized in a series of cases that a pharyngitis came on first—a postnasal condition—and then an attack of appendicitis. He believed that it was "rheumatism" of the appendix. Dr. Lyman recognized the sore throat in connection with the appendicitis. I remember the first case I saw in consultation with him, because I was so positive on one side and he was equally positive on the other, and we were both right. The patient was a child. He had pneumonia when he came in with his appendicitis. We did not operate on him. If you operate for appendicitis in the presence of pneumonia you usually have to sign a death certificate

afterward as the reward for your trouble. We tided him along until his pneumonia disappeared; then we operated.

This patient has been operated on before, which was not mentioned in the history. A drain was inserted. I am going on in the line of the first incision, opening the abdomen in the usual way. The omentum is adherent. We will separate that adhesion.

There is the cicatricial tissue all along the line of the drainage-tube. All the intestine is adherent at this point. We must carefully liberate these adhesions before we will be able to expose the appendix.

That done, the caput coli rolls up into the field. We have the third degree of obstruction around the base of the appendix, and there we will have organic adhesions. There is the ileum. There are the adhesions from the ileum down onto the base of the appendix. I will free these adhesions and see if we have any part of that appendix left. I can see where the abscess formed. There is gray granulation tissue around the site of the abscess. That is the result of the infection. The gray mass is the degenerating product of infection getting ready for absorption. I can feel the thickened mass where this abscess burrowed down into the neighborhood of the great mesentery.

I will free this mesenteric adhesion. These adhesions are not organic; they are not fixed. Here is the ileum and the ileocecal valve, and here is where the appendix is normally located. I am trying to feel anything that I might recognize as the stump of the appendix. I cannot see or feel anything like an appendix attached to the caput coli where the appendix ought to be. I will free these adhesions further back and see whether the distal end of the appendix is here or not. The proximal end appears to have sloughed off from the bowel. I can feel something like an appendix. Here it is, and I am rather fortunate to find it. There is only the tip left. The appendix has sloughed. We will free that from its mesentery and I will be able to bring it up. All the distal portion has sloughed off. This girl would probably have had recurrent attacks of inflammation in the stump, but she never could have

had a typical appendicitis. We advised her to go home, but she felt if she went home the attack would come on again, and she decided to have the operation done at this time.

We will put in the puckering-string suture in the usual way and ligate the vessel of the mesentery.

We will conclude the operation in the regular way and remove the remnant of the appendix.

This case illustrates so many points in connection with an operation at this stage of the disease, and particularly the process of absorption of the adhesions. Now we put in the regular Lembert stitch and cover up all that line of union. The danger of infection in this operation is very much less at this stage of the disease.

We will close the peritoneum with a continuous catgut stitch by everting the edges, and then suture the abdominal muscles carefully so as to forestall the possibility of a ventral hernia occurring later. Figure-of-8 sutures close the fascial incision; and horsehair approximates the skin-edges. We do not put in a dressing. A gauze collodion dressing seals the wound, thus guarding against infection from without. [The patient made an uneventful recovery.—Ed.]

COLONIC ADHESIONS SIMULATING RECURRENT APPENDICITIS

HISTORY

FEMALE, teacher, twenty-one years old, entered Mercy Hospital because of recurring attacks of pain in right iliac region. Her menses have always been regular, and she has always been free from pain. A slight leukorrhea has been present for the past eighteen months. She has never been married and never missed a menstrual period. Her life has been free from serious illnesses. She never has had typhoid or other intestinal disorders.

The first abdominal pain this girl ever experienced was in the summer of 1906. It was in the right iliac region. It never became severe and caused her inconvenience only for one day. In the fall of 1906, three months after the first attack of pain, she was seized by a sharp, colicky pain in the right side of the lower abdomen. It was quite severe, but not sufficient to cause her to leave her class-room, and persisted only for one day. During 1907–08–09 she remembers having had attacks of similar pain at intervals of from one week to two months. These never kept her out of school, and the pain always passed off after one or sometimes two days. She never became nauseated, never vomited or had a fever at these times, as far as she remembers. Between attacks her health was perfect. She never had eructations of gas or a heavy sensation, as of fulness, in epigastrium after eating.

Since 1909 the pain has been more severe. It has recurred nearly every two or three weeks. Seven times she has been kept in bed for three or four days at a time with very severe pain, always at McBurney's point, but often radiating to the center of the abdomen or to the right lumbar region of the back.

At these attacks she has always been nauseated and had emesis and has often felt feverish.

The present attack began Saturday, as usual, with severe, colicky pains at McBurney's point. She went to bed immediately and has stayed there ever since. Two hours later she vomited. She had no chill nor has she ever had one. She is sure she had a fever. She noticed, as she has often done during these attacks, that the muscles of the right lower abdomen seemed rigid and that a swelling the size of an egg rose up at the site of the pain. This was directly over McBurney's point. The pain has subsided and recurs only if she turns quickly in bed. A tenderness still exists. Temperature on entering hospital, 99.8° F. She has a slight cough. Heart and lungs normal. Slight leukocytosis.

COMMENTS AND OPERATION

DR. MURPHY: What about those last attacks, doctor?

THE INTERN: She was nauseated.

DR. MURPHY: She was incapacitated for three or four days?

THE INTERN: Yes.

DR. MURPHY: Was her temperature taken at any time?

THE INTERN: No, doctor. [Reading further.]

DR. MURPHY: Swelling comes on only at the site of the pain? When does it come on?

THE INTERN: After each attack.

DR. MURPHY: After each attack. What do you mean? "After" is a very indefinite time.

THE INTERN: This is not my history, doctor.

DR. MURPHY: It does not make any difference whose history it is. Does she get that pain and then does she get elevation of temperature, nausea, and vomiting? Does all that occur within a few days? When there are fixation and binding of the ascending colon by these pericolonic bands, and in these cases this produces temporary obstruction and distention, with enormously dilated caput coli, hypertrophy of the caput coli, with sensitiveness during the period the tension is on, then rapidly subsides and disappears until another attack comes on. So classic is that

condition that you can make a diagnosis on this circumscribed condition or on this sensitiveness to pressure and on the fact that when the patient commences to get well he will tell you of a rumbling of gas, an explosion, and the attack is over. When you analyze these cases you will be surprised how accurately they describe that condition.

That is not recurrent appendicitis at all; it is recurrent retention and distention of the caput coli and is classic, and many of these patients are operated on as appendiceal cases, with no attention paid as to the facts, with the result that the patient comes back again and again, and goes from one doctor to another seeking relief. The kidney is tied up, the appendix taken out, a tube taken out and probably an ovary, and all the time the diagnosis is recurrent appendicitis. It is only when we make a careful analysis of these cases that we can make a differential diagnosis. One operation after another is done on the right side of the abdomen, and, as the Mayos would say, all the movable organs are fixed and all the fixed organs are made movable, and finally all those that can be removed are removed, and still the patient is not relieved of the pain and other symptoms.

DR. MURPHY (To intern): What was the leukocyte count when she came in, doctor?

THE INTERN: 5800; the count yesterday was 9000.

DR. MURPHY: Was a tuberculin test made?

THE INTERN: No, doctor.

DR. MURPHY: It should have been made. Why? Because she had a leukopenia when she came in. Either that count is incorrect or there is something wrong somewhere. It does not fit. She had no soreness when she came here. She came with an acute exacerbation the day before yesterday, and then had a leukocyte count of 9000 yesterday; you see how one little feature puts things all out of gear. She had an acute attack, and with this attack she had a leukocyte count of 9000. Put that in the record. The recurrent attacks were not of the type that we see so commonly in tuberculosis in the abdomen. We had a beautiful case a week ago today. The case was sent in as an

appendicitis, but before we opened the abdomen we said it was not appendicitis, because the clinical history did not fit. We said that it was probably tuberculosis or some other complication. This woman had tuberculosis. We palpated a tuberculous mass running clear across the abdomen. Furthermore, it was not a tuberculosis of her omentum, but a tuberculosis of her mesocolon. There was a tuberculous ulcer in her colon. That is one of the reasons why a tuberculin test should have been made in this case. In that case we made the test and had a negative response. This is the third case of tuberculosis in the intestine that gave a negative response to the tuberculin test with a pronounced tuberculosis present.

We will make the usual incision for removing the appendix. We can lengthen it later if necessary. I insert my finger, and endeavor to pick up the caput coli. There is no Lane kink. There is the appendix. It is not the seat of an acute inflammatory condition. It is curled in at the under portion and knotted on itself, but it does not show evidence of an infection which would cause this elevation of temperature. We will remove it and have it examined by the microscopist to see whether it is a case of retention with absorption sufficient to give that leukocytosis. We do not believe it is. It does not look so.

There is a secondary mesentery present. That is an interesting manifestation. We have had a number of them recently. There is the mesentery; I pass my needle through that entirely and clear across so as to take up the peritoneum on both sides and then come back and catch the peritoneum on both sides, so that when I pull that suture taut I will have what? A complete circle of peritoneum. You will remember Wölffler found that was his weak point in making his gastro-enterostomies. I am getting this additional mesentery out of the field. Now we are ready for the amputation of the appendix.

One of the things I want to call your attention to in this case is the pathology on the surface of that caput coli. You see that vascularity all over the surface of the caput coli. That is pathology. I have not found the etiology of that condition, but the etiology is there because that vascularity is a sequence. It

never occurs without a cause. We will put a ligature on the appendix. There can be no bleeding into the bowel from that. First, get a ligature around the appendix; second, a puckering string in the caput coli which to all intents and purposes is a Lembert stitch, and, third, a final Lembert stitch. This is the way I would close a bullet wound, and I am as careful and cautious in putting in this suture here as in closing a bullet wound. In following that plan we have had good fortune. We never operate in a case of tuberculosis that involves the base of the appendix. We never take it out.

Let me see why we have this vascularization. It is not from any lesion of the appendix. There is no evidence of tuberculosis in the ileum. No evidence of any lesion there. The vascularization shows that we have some type of inflammation in the neighborhood, either within or without the bowel. Let us examine this caput coli and see if there is tuberculosis present there. Here are some fixations extending upward; before I touch that I am going to return this intestine into the abdominal cavity and see if there is any trouble in the pelvis. We will examine, first of all, the most common site of trouble in the female, namely, the tube. The tube is not distended. It is a little congested, but there is no evidence of tuberculosis. There is some fluid in the pelvis. There is an infection, a submucous infection, somewhere. There is some thickening of the tube, but not enough for tuberculosis. This is not a tuberculosis of the tube. That is out of consideration. We will see if there is tuberculosis of the sigmoid. The transverse colon is away down in the pelvis. It is not the sigmoid. I am going to try and bring the sigmoid into the field. I can feel the uterus and the bladder and the tube on the opposite side, and a small cyst of Morgagni. That is of no pathologic significance. The other tube is not involved.

Now let us see why she had that recurrent pain in the upper abdomen and see if we can detect fixation or adhesions. Now you see how nicely we get on with this incision without cutting the muscles. There is some fixation due to adhesions at the hepatic flexure between the colon and the parietal peritoneum.

That is the cause of the repeatedly recurring retention. The gall-bladder can readily be brought into the field. It is normal. The pyloric zone of the stomach and the duodenum are also normal. The pylorus is not distended, nor is it thickened. It is perfectly pliable and soft. There is no white line or ulcer on the anterior surface. It is perfectly clear.

Here is the etiologic factor. These colonic adhesions come from the outer side of the abdominal wall. I do not know that we have accurately explained the etiology of these cases. We have been having considerable discussion about it lately, but to me it is not clear whether there has been an excessive irritation here. I will divide the peritoneum so as to get that piece out and let that bowel go down.

What about the constipation phase of this case, doctor? it has passed out of my mind.

THE INTERN: Her bowels were regular, doctor.

DR. MURPHY: There is the caput coli. See where this bowel was suspended! We put the carbolated gauze on these surfaces so that they will not re-attach.

Let me say again, with emphasis, that when you find that the physical findings and the analysis do not fit there is something wrong and you must make a more careful investigation. It is easy to make a diagnosis of recurrent appendicitis. That is a very common error.

Let the record show that on opening the abdomen the caput coli was easily drawn up, and that on its peritoneal surface it showed an area of vascular distention and connective-tissue development. The transverse colon was found in the pelvis. There was no tuberculosis there, and while there was some slight involvement of the tip of the appendix, it was not enough to make it pathologic. The right tube and ovary were then brought up into the field and inspected. There was no tuberculosis of the tube, nor was there congestion or enlargement of the vessels around the tube. The left tube and ovary and sigmoid were then brought over and showed no evidence of disease. The uterus was not surrounded by adhesions and was easily palpated. There was a small Morgagni cyst in the left mesovarium. It

was found that the trouble in this case was due to fixation and adhesions at a level with the lower margin of the liver coming from the outer parietal peritoneum and suspending the colon and caput coli. These adhesions extended down to the caput coli like a network, and hung over and suspended it.

Let the record show further that the gall-bladder was examined and there were no stones; that the duodenum showed no evidence of disease, and there was no evidence of a gastric ulcer.

We have done all that is necessary in this case, and will proceed with the closure of the abdomen. The peritoneum is closed with a continuous catgut suture, everting the edges, making an ectropion, so that there will be no raw surface in the peritoneal cavity. That will prevent the formation of adhesions. Next we insert figure-of-8 stitches of silkworm-gut, completely obliterating all dead spaces.

The suggestion of Dr. Gerster, of New York, was very timely when, in connection with this class of cases, we were discussing the reason for failure in results in operations on the gall-bladder. One of the reasons to which he attributed poor results was that the incision was too small, and in our efforts for getting beautiful cosmetic results around the appendix we have in these chronic cases made our incisions too small and by doing so hazarded the patient's life. In the other cases in this class we hazarded the finding of the true etiology of the condition. We need larger incisions and more careful exploration after the abdomen is opened, and we need space in which to do that.

The skin-edges are approximated with horsehair, making a continuous suture. The wound is then dusted with bismuth subiodid, a desiccating powder, not an antiseptic, and sealed with a collodion gauze dressing. [This patient made an uneventful recovery, and at the time of leaving the hospital was free from all symptoms.—Ed.]

EXOPHTHALMIC GOITER

HISTORY

FEMALE, single, twenty-one years of age, entered Mercy Hospital complaining of an enlargement at the site of the thyroid, palpitation of the heart, and nervous exhaustion. Four years ago she had typhoid, but otherwise has been free from serious illnesses until present trouble began.

In the spring of 1910 she noticed a slight enlargement just beneath the middle of the right sternocleidomastoid muscle. It increased steadily in size, and six months later she noticed a swelling in the midline and also somewhat on the left side of the neck. During the latter half of 1910 she worked hard. She felt that her strength was less than usual, and she became tired and nervous readily. When she danced or otherwise exercised she noticed that her heart would beat fast and forcibly, much more so than the amount of exertion would justify. In January, 1911, she was excitable and nervous. During the latter part of that month she began to have very severe headaches and felt exhausted. At this time she first noticed that the eyes were more prominent than usual. This sign increased in degree.

During February, March, and April of 1911 she was forced to stay in bed because of nervous prostration. At this time she often felt her heart beat forcibly, and she also had difficulty in breathing. She had intense headaches and very severe and prolonged attacks of vomiting, lasting often three to five days. She became emaciated, and could keep nothing on her stomach. She was placed on thyroid medication and kept quiet in bed. In June, 1911, she was able to be up and about, but suffered from nervousness and palpitation of the heart on the least excitement. Her condition has been about the same since then. It seems to her that the enlargement in the neck varies in size at times. Its growth, however, has been fairly constant, and it seems to be

becoming of a firmer consistence. It never has been hard, nodular, or fixed to the surrounding tissues. Pulse ranges from 98 to 124. Urine is normal. Blood findings: Erythrocytes, 4,200,000; hemoglobin, 80 per cent.; leukocytes, 9,800; differential count: Polymorphonuclears, 52 per cent.; small mononuclears, 41 per cent.; large mononuclears, 3 per cent.; transitionals, 4 per cent. No eosinophils were found in a count of 200 cells. Blood pressure, 85 mm. of mercury.

COMMENTS AND OPERATION

Dr. Murphy: This is a fairly classic history of this lesion. There is a very definite relation between the clinical history and the pathologic findings in the thyroid. You can divide lesions of the thyroid into three grand classes: first, the lesion of the epithelial or secreting cellular elements of the gland; second, the lesion involving principally the blood-vessels; and, third, the lesion involving the connective tissue. So that there are three classes of lesions—lesions of the connective tissue, the lesions of metastatic infections with a hyperplasia of the connective tissue in the gland, with also some increase in the epithelial elements, pain and swelling, coming on usually as metastases from some other infectious disease, such as influenza, typhoid, and scarlet fever. This acute swelling continues usually for some weeks, then gradually subsides. If the inflammatory process is long in duration and severe, the connective tissue commences to contract. It can entirely obliterate the secreting cells in the gland and cause hypothyroidism.

I had two very striking cases of that class, one following tonsillitis, with an acute infection of the thyroid, edema, swelling, and sensitiveness. This patient I treated years ago, when she was a girl, for an enlarged thyroid. Later, after she was married, she developed some of the mild symptoms of Graves' disease, but under the arsenic treatment they disappeared entirely. About two years after that she was confined, and a year after the confinement she had a sudden severe tonsillitis which caused swelling and soreness in the lower part of her neck. She had an intense toxic nephritis, with blood in the urine, uremia with

convulsions, unconsciousness, and blindness. From this she was relieved by an enormous blood-letting. This went off into a subacute condition, and about a year after the onset the edema and swelling subsided so that the woman fell off in weight from 215 or 230 pounds to 165 pounds. She had put on a great deal of adipose tissue within the three years preceding this metastatic infection; then, as her albumin diminished (but never disappeared), she had a second attack of edema and swelling of her limbs, and when I saw her she looked like a mass of blubber, filling a three-quarter-size bed and weighing over 300 pounds. The case puzzled me. Microscopic findings in the urine did not justify the conclusion that that colossal edema of her legs, arms, and abdomen was from her nephritis. She had a weekly or ten-day explosion of high temperature, and then it subsided again until she got another attack. That condition continued to recur. She had pain in the abdomen, with a profuse diarrhea. I saw her in consultation during my spring vacation, and it was two weeks before I arrived at a diagnosis, watching her every day and looking her over. Suddenly it dawned on me—what had become of her thyroid that had always been enlarged? Notwithstanding the swelling in her neck, I could determine that there was no thyroid. Then I said, this is a myxedema. Her eyebrows had almost disappeared, her hair, which had formerly been heavy, was just a wisp; her axillary hair was gone; her pubic hair was gone. This falling out of the hair was the key to the secret; it is a part of the general symptoms that go with myxedema. So I made a clean-cut diagnosis of myxedema. I put her on thyroid extract and she immediately began to improve and went down from 300 pounds to 120 pounds. She still had some albumin and some casts in her urine, but she got perfectly well. She again took up her social interests; she went to California the following winter and participated in everything that was going on, early and late. Suddenly she developed an acute septic endocarditis, then a pericarditis, and died in about three weeks.

Another patient was sent to me from Nebraska a number of years ago with much the same type of attack. I made a

diagnosis of acute myxedema about six weeks after the subsidence of the swelling of her thyroid.

These are the acute manifestations, with the subsequent history of the lesions of the connective tissue.

There is another lesion of the connective tissue of the thyroid and that is the sarcomatous disease. Not infrequently sarcoma originates in the connective tissue of the thyroid.

We know that we can produce all the symptoms of Graves' disease, excepting the exophthalmos, by the administration of sheep's thyroid. They get the typical tremor and palpitation of the heart—all the symptoms of hyperthyroidism.

This hyperdevelopment of the vascular system is the principal lesion that occurs in connection with the vessels of the thyroid.

The important lesion—the common lesion—is a lesion of the secreting structures or epithelial elements and ducts of the thyroid. These lesions are divided into, first, the simple hyperplasia of the gland, an increase in the acini and an increase in the number of cells lining the acini. Second, a circumscribed increase, with an encapsulation, causing what is known as an adenoma of the thyroid. This is identical with the first lesion except that it is circumscribed and encapsulated. Third, multiple adenoma. Fourth, an increase in the secretion of the cells without absorption; in other words, an increase in the colloid material. When you cut into the gland you find a large mass of colloid material. That is the colloid goiter, which can be divided into the diffuse and circumscribed, and finally there is the combination of these two conditions.

We will operate on this patient now. We will make the so-called collar incision, extending across the neck from one sternocleidomastoid muscle to the other, and passing over the center of the enlarged thyroid. A crease in the skin is selected, so that the resulting scar will be less conspicuous. The incision extends through the skin, fascia, and platysma. The important thing in all these cases is to get, first, a good exposure, and, second, to have enough hemostats and have them on at the right time. The sternohyoid muscles are now exposed and must be divided so as to give us better access to the gland. I pinch the

muscles with a hemostat before dividing them so as to minimize the bleeding. We divide these muscles high up close to the margin of the sternocleidomastoid, which we will also divide for a short distance. Now we have a splendid exposure of the thyroid.

Let the record show that this girl was very hoarse before the operation. I want that made a part of the record, because for some reason her recurrent laryngeal nerve was interfered with and we could not determine just what that reason was.

Now I commence to wipe off the tissues on the inferior margin of this gland; and now I come close on to the zone in which the recurrent laryngeal nerve is situated. Here is the inferior thyroid. The recurrent laryngeal nerve is in close proximity to that. There is a parathyroid! See that little yellow spot right there! Now the lower pole is fairly well loosened and we turn our attention to the upper pole. It is high up. We provided for that by extending the incision on that side away above the average position.

The recurrent laryngeal nerve is visible. There is no traction on the trachea. We are keeping close to the capsule of the gland all the time. That is the principal and only precaution that we can take to protect that recurrent laryngeal nerve. We are going to remove all of one lobe and a portion of the other lobe. I now incise the capsule of the gland. Now we will have use for many hemostats because we wish to have as little hemorrhage as possible. As fast as we grasp a portion of the gland with a hemostat we excise it. After we have removed the desired amount of gland tissue we will ligate every bit of the stump in the grasp of these hemostats with catgut. Before suturing the capsule over the stump we will search carefully for bleeding points and ligate any that we may find. Having done so, we are now ready to suture the cut ends of the muscles together, using catgut.

I am sewing up the sternohyoid muscles just where I divided them, and also the nick in the sternocleidomastoid. Deep catgut and superficial horsehair sutures approximate the wound edges without tension. A gutta-percha tissue drain is passed

down into the lateral angles of the wound and emerges in the midline.

The other day I made a ligation under a local analgesia. That night the boy had a temperature of 103° F., notwithstanding the small incision, and a pulse of 148. I merely ligated the upper pole. His has been a desperate case. We had him here for a number of weeks getting him ready for an operation. He had a little diarrhea; his pulse went to 160, and he almost died. These patients do not stand anything; you are afraid to touch this class of cases. You get them on the borderline, just ready to die. We have them come in from the country in the early fall, never touch the patient, and he dies in a week. We keep them still, apply the ice-bag, and do everything else necessary, and still they die, showing how desperate these cases are. This is the class of cases for which there is no control; if you give them adrenalin they get intensely intoxicated and they may die. Give them a little morphin if very restless—that helps them.

Now we have the normal conformation of the neck restored. The wound is sealed with gauze saturated with collodion, and a large dressing is applied to absorb the wound secretions.

Now let us go back to the pathology of this case.

I was speaking of the excessive secretion in the cells and acini. Where this is still more circumscribed and where you have much secretion without absorption in the circumscribed area, you may have, as a sequence, a circumscribed cyst. This cyst first contains colloid material, or the normal secretion from the cells of that particular acinus. As the quantity of secretion increases it produces a pressure on the cells, causing them to flatten. With change of conformation of the cells comes a change in the character of the secretion. The secretion rapidly ceases to be normal colloid material, but becomes serous material. When these cysts are opened they are found to be monolocular, rarely multilocular. You may have two cavities contacting with each other, but not communicating. That is in conformation with the law of change of secretion in every epithelial secreting gland. As the change in the cell conformation takes from the cell all its normal physiologic secretion, it gives it the character of a

serous secretion in place of an epithelial or glandular secretion. Occasionally there is a hemorrhage into this cyst and there develops what is known as a grumous cyst of the thyroid. These are only changes in the product and not changes in the tissues themselves at all. The most common position is in the isthmus, with its tip in the gland.

I remember a doctor who had one of these cysts, and he almost suffocated before he got to the hospital, where the late Dr. Alexander Hugh Ferguson was operating. Dr. Ferguson opened the cyst, that was all that was necessary to do, and drained it. It contained grumous material. The process of repair went on, and cicatrization occurred. That was a cure as well as a relief of the danger to the man's life.

The type we have under consideration now is the increase in the colloid material, and an increase, as we believe, only of a physiologic product, having in this particular individual a very deleterious effect on the central nervous system.

The Graves' disease syndrome of symptoms is always recognized. There is one time, however, to recognize it, and that is long before the exophthalmos appears, and long before the gland is large enough to be seen by everybody in the block except the doctor, and long before the patient has a grave intoxication of the central nervous system, and long before the heart is impaired so seriously that it is a very dangerous thing to do anything with the patient. It is not necessary for us at this day to watch for all these grave manifestations before we recognize and before we treat the disease. I think it can safely be said that Graves' disease, from its very incipiency, is a surgical disease, and that our hope in the future in Graves' disease is going to depend on timely operation, and we are going to be responsible for acting in time. The results in the treatment of Graves' disease through surgical management are illustrated in the work of Charles Mayo and Theodore Kocher, and of many others who have operated on the thyroid with splendid success. So that there is no longer any question as to the result of surgical treatment. We know, from a clinical experience running over a quarter of a century or more, that there is no question as to what the previous results were with

medical treatment, so that the surgical treatment is the only treatment that is indicated in this disease, but it must be timely. I think this is one of the diseases where the surgeon should not touch the late case. When they get to that serious condition, they must not be operated. Put them in bed, elevate the foot of the bed, put on the ice-bag, and give them sodium cacodylate. Keep them absolutely still. That does not mean get up half a day or have company half a day, or read correspondence, or get up and go to the toilet—nothing of the sort. Just as in the case of this boy we have here now, carry them along and you get to the stage where you can do one procedure after another.

I recall a patient who came to see me in what I consider was the early stage of the disease—about six weeks after the initial manifestations. The girl had learned of a serious breach of family obligation on the part of her father, and it depressed her nervous system so that in two weeks she had an enormous pulsating goiter, heart-beat 160 to 180. To touch that girl at that stage would have been fatal. We put her in bed, and put an ice-bag on. The family doctor who had charge of her says he carried out the instructions to the letter. In fourteen months that gland was contracted, heart-beat down to 80, and she is back again to her normal condition, with a fairly firm and somewhat enlarged thyroid gland. The firmness of that gland is due to the cicatricial contraction around the acini, which were enlarged.

A sudden contraction after a gland has been large is a serious matter. I can illustrate the seriousness of that situation by citing a case. The patient came over from Michigan with an enormously enlarged gland and in quite a serious condition. We kept her for three months on the ice-bag and rest treatment. She improved so that we could perform a ligation of both the upper poles. She improved rather rapidly and went home. That was in February. When I returned in August I had a letter from her saying she was not well, and that she was coming to Chicago. The gland was found to be still large, and just as hard as a piece of iron. This started up a thrombosis in the vessels in the gland. Then it began to contract, which made it

so firm and hard, obliterating all the secretion from that gland, destroying it as a secreting gland, and she developed a myxedema and died within a week after her arrival. That shows you one of the dangers of contraction of that connective tissue, or thrombosis with subsequent contraction and obliteration.

In this variety of goiter, therefore, you have to take into consideration when you look at the gland, first, which of the three tissue elements are involved; second, if they are involved, are you having the more serious stages of Graves' disease, and is there produced more thyroid secretion than that system can care for?

In the treatment of hyperthyroidism many questions have come up as to the medical treatment. I have seen 100 or more of these patients get perfectly well without surgical intervention, and I have seen a number of patients with Graves' disease get well without surgical intervention. With rest, ice-bag, arsenic, and no mental irritation they practically all get well if they are of the typical hyperplastic type. That class of patient gets well on medical treatment. That is a class of cases that is absolutely medical in the beginning. There are patients who get tremor when they straighten out the fingers, which is one of the very early signs; they get tachycardia long before they get exophthalmos. If they get that, operate then, before the heart muscle cells are thick. Operation in the early stages does not cause a mortality of 0.5 per cent. It is one of the safest operations in surgery. Operate in the late stage of Graves' disease, and it is one of the most dangerous operations. You are absolutely at the mercy of the patient and the intoxication.

TRAUMATIC LESION OF BRAIN

HISTORY

THE previous history of this patient is of no bearing on the lesion for which he now comes to us.

On March 14, 1912, about two months ago, he was struck by a fellow laborer with a tamping pick over the right postparietal region accidentally. He fell to the ground and was unconscious for a minute (?). When he recovered consciousness, he was weak and nauseated, but did not vomit. He bled profusely from the scalp wound, but did not bleed from ears, nose, or mouth. The doctor who was called sutured and dressed the scalp wound, but did not explore the skull beneath for evidence of depression or fracture. There was primary union in the scalp wound.

The patient was not put to bed, but has been up and about every day since the injury. About a week after the injury he developed occipital headaches of a throbbing character. The pains radiated from the right occipital region down the neck. When the headaches get very severe, the patient is unable to hold his head erect. No motor or sensory disturbances, except a numbness in the left index-finger. This, however, is not continuous. No double vision; no loss of memory; patient answers questions intelligently; some vertigo and there is a sense of pressure over injured area; reflexes normal.

COMMENT AND OPERATION

This is a typical history, with a few exceptions. The principal exception is the speed of onset of the symptoms. In traumas of the skull, even with fractures that are demonstrable, the rule is for the patient not to have the headaches, the vertigo, or the sensory or motor disturbances so soon after the injury unless they occur instantly from a trauma of the cortical portion. They

usually come on many months and sometimes years after the accident, and the later they are in coming on, the more grave and serious they are.

Management of skull injuries—of the concussions that render patients unconscious, of the contusions with depression of the skull, and of the depressed fractures of the skull, and of fractures of the skull at the base—is one of the dark chapters in surgery,—dark so far as the *ultimate* outcome is concerned,—and one of the bright chapters in surgery so far as the *immediate* effect is concerned. That is, patients may have very severe injuries,—compound fractures and comminuted fractures, with the escape of large quantities of brain tissue, with the skull fragments driven down into the brain for an inch and a half (which are removed),—and the patient may recover immediately from the accident and remain well for a long period of time.

The injuries with compound or depressed fractures are the least injurious; that is, where the edge of a sharp instrument penetrates the brain, like the claw of a hammer, a nail, or scissors, if the patients recover from the immediate effect—and they do with proper treatment—you rarely have any trouble subsequently. But the blows which are sufficient to produce unconsciousness, with or without a fracture, never leave the patient free of the danger of a cerebral complication or a nerve complication for all time. The period of time that may elapse before the serious symptoms come on varies greatly. The serious symptoms sometimes come on within a few weeks, or a few months, and again not for many years. I remember one man who had his first convulsion twenty-seven years after he was kicked on the head by a horse. He had a depressed fracture, compound, which was elevated at the time and healed. Many years ago I had a patient who fell from a second-story window. The child was unconscious for three weeks; it had a comminuted fracture of the skull. The child got well, developed into a man, advanced to a very prominent position in a bank, and twenty-two years after the accident he had his first convulsion. Then he had convulsions every few weeks, and he is now completely incapacitated by his convulsions.

I cite these cases in order to impress on you the teaching of the late Dr. Fenger concerning the management of skull injuries, which should be a more general practice with the medical profession. Fenger realized, I think more than any other man, the significance of a blow of any kind on the head that rendered the patient unconscious, with or without a fracture. It was Fenger's teaching, when I was an intern in the Cook County Hospital, that every man who came to the County Hospital who reported that he had been injured sufficiently to render him unconscious should be put to bed with an ice-bag on his head and kept in bed six weeks, regardless of what his symptoms were, so as to give his cerebral tissue and his meninges an opportunity to return to the normal with the least deposit of connective tissue. Fenger impressed on us the idea that these cases many years later would manifest symptoms, and that if we hoped to avoid these symptoms, they were not to be avoided so much by the operation as by the quiet treatment which these patients were to have immediately after the injury.

We have no idea as to whether this pick went through this man's skull and whether it injured the meninges as a direct result of the trauma. We do not know. We do know, however, that this man has pronounced signs of local irritation, either from the trauma, from the direct penetration, or from the concussion, and we also know that these headaches, once they start, usually lead to very serious manifestations. The most pronounced symptoms are, first, the recurrent headaches; after the recurrent headaches have continued for months or years come the attacks or convulsions, and then the grave attacks of epilepsy, and once the patient has that type of epilepsy, he is practically doomed. In a large experience we have had only one case in twenty years where a cure followed operation in a grave case of epilepsy. We have had some very strikingly good results, and we have had a great many very lamentable failures, but in talking to these patients when the epilepsy has come on I say to them plainly, "Your chance for recovery by doing an operation on your brain is not very promising."

The type of operation varies. Originally we merely took out

the irritating spicule of bone. We just took out the depressed fragments of bone, and if we did not find them, we would be discouraged. In later years we learned that removing the depressed fragment of bone has little or no effect on the symptomatology in these cases. We now take out an area of dura that has been traumatized and that has given us the most pronounced relief.

I recall an incident that occurred when I was a student in Rush Medical College. A French physiologist came to America making certain demonstrations regarding irritations of the dura, and one was that in a pigeon he could produce epileptic convulsions by the slightest electric irritation of the dura, and it has always been my belief that it is the dura that is the irritating zone. It is my belief that the connective-tissue deposit in the dura produces a type of irritation that causes the convulsions. That is why, in cases of fractures of the base with convulsions, we never do an operation because we cannot remove the dura in that zone.

[To Assistant.]—Where has this patient been marked? We do not want to take out the wrong side, an accident which is very frequent. Remember, the wrong eye has been taken out, the wrong leg has been amputated, the wrong Gasserian ganglion has been removed, and the wrong side has been operated on for hernia because of a lack of system in marking. *If you have a system, that is what counts and saves trouble.*

The head has been shaved and painted with tincture of iodin. A rubber constrictor has been placed around the head just above the ears. We wish to locate, first, the Rolandic fissure. Take half the distance between the occipital protuberance and the base of the nasal process of the frontal bone, add half an inch backward. That is the line. Just back of the Rolandic zone.

That zone will form the center of our flap, which will be nearly circular, base down, measuring about two inches at the base and four inches in its widest diameter. The only evidence of the injury is a very slight scar, barely visible. His veins are dilated considerably, and that is where the bleeding we see comes from. The slightest pressure on the skull stops it.

I recall a case that came to me from Oshkosh, Wis. The patient had been injured eleven years before, as I remember it now. He was sleigh-riding, the horse ran away, the cutter tipped over, and his head collided with a telegraph pole. He was unconscious for four or five days. Then he apparently got well and went along nicely. He went to school; had the usual work. Some years afterward he began having intermittent headaches. They became more and more severe. Finally they became so severe that he had to be taken out of school.

We first operated on him by removing a small depression that existed, with a cyst under the depression. He improved and remained fairly well for I forget how many months, when he commenced to have headaches again. One day he had three convulsions, became unconscious, and remained unconscious. My associate at that time, Dr. Lemke, went to his home and saw him. He drilled through his skull, tapped his lateral ventricles, which relieved the internal pressure, and he was better for the time being. He remained well for a few weeks, when he had another convulsion. They brought him to Chicago, and I operated on him for the third time. In the second operation we removed a large area of the dura. We made an incision something on the type I am making now, and removed a large area of the dura. He recovered, and when I heard of him some years afterward he had neither a return of the headaches nor of the convulsions. That is a striking contrast between the drainage of the ventricles on the one side, the removal of the spicule of bone on the other, and the removal of the dura, on the third.

Since that time I have made more extensive removals of the dura than we ever did before, and since that time I can say that my results have been improving, but they have never been gratifying—they have never given results like those obtained in other phases of surgery. Of course, you must admit in the beginning that no one knows yet what the true etiologic factor of epilepsy is, and our chances of bettering the situation very much are not great until we get a more precise knowledge of what

epilepsy is. We know some of the conditions that produce it—that is, a few.

Having turned down this flap of muscle fascia and skin, we will make a trephine opening and then chip through the skull to make the bone-flap. This is a very thick skull in this neighborhood. For everyday work the DeVilbiss instrument is the best; for extensive work, the Neff instrument is the best.

We have completely outlined the bone-flap and shall turn it down by fracturing it at its base, without detaching or removing its blood supply. We always make an osteoplastic flap. I do not have to cut the scalp. I leave it intact. I crease the bone in the direction in which I wish to have it fracture. One must be careful not to penetrate the dura. Now I will free the adherent dura from the bone-flap.

Let the records show that the dura bleeds profusely everywhere; in place of the ordinary free white membrane one sees when the dura is normal, see how it bleeds, showing the inflammatory condition that has been present here. Probably as the result of the trauma the dura was separated from the bone and has been developing new vessels for the purpose of reattaching itself to the bone. You see the pick did not come through the skull. There is no evidence of any injury of the inner table of the skull where he was struck, showing that the effect produced on the inside was a concussion effect and not a penetrating effect.

You will remember the splendid paper by Dudley Allen, of Cleveland, written some years ago, in which he analyzed a large number of cases of skull injury without penetration of the dura and most of the patients eventually found their way into an asylum. Many of them had subdural irritation.

I want to take out a certain zone of that dura, but I must leave enough dura not to overlap the margin of that bone-flap when it comes back, otherwise the flap will sink down. Note the thickness of that dura. It is at least three times as thick as the normal dura. Of course, that is in consonance with this finding on the outside. The pia is also thickened. The cortex beneath is edematous right under where the pick struck. The edematous area is very evident and is highly edematous.

That is one of the prettiest things I ever saw in all my head surgery. Beyond the zone of reaction the dura is normal. There is an inflammatory condition around the vascular network, with an exudate. He evidently had a hematoma beneath and an inflammatory condition. There was no evidence of a depression, nothing when I lifted the skull, except this extreme vascularity, which attracted my attention. Formerly, if we found no depression we let the dura alone.

I will excise the thickened portion, thus removing the focus of irritation. We will not go beyond the line of the osteoplastic flap, because if we did, the dura would again become adherent. We will leave a rim of dura all the way around on which the flap can rest and not sink in. I want to put in a piece of paraffin to prevent adhesions from forming. A thin piece of paraffin will answer the purpose. Now I have the dura free. That piece of paraffin will stay in there and it will become fixed. It covers all the brain surface which we denuded of its dura.

Now we can see the reason for all this man's symptoms, even in the gross pathology demonstrable.

Visiting Surgeon: What is the length of time before that paraffin will disappear?

Dr. Murphy: It will never disappear. This is a coal-tar paraffin. If it were a whale-oil paraffin, it would become absorbed. Mineral paraffin is not absorbable at all. We have often had to remove it when it was used for correcting facial deformities. Then, again, we have had to remove it many times years afterward when operating for the cure of a hernia where paraffin had been injected into the abdominal wall over the internal ring. There was no effort at absorption; the paraffin became encapsulated. And the capsule around it looks like a serous membrane—smooth and glistening. That has been one of the things that led me to consider a plan which I am going to put into execution when I get to the first joint where I cannot get sufficient fat and fascia to cover the articular ends of the bone. I am going to cover the raw end of the bone with paraffin —just a very thin plate of paraffin—and let it stay. If the paraffin were strong enough to carry the weight, we would put

it between the bone-ends to prevent union. It is never absorbed; the granulation tissues do not grow into it, and it would insure earlier mobility and it would be very much less painful than the plan we follow now. It is like the transplantation of fascia flaps taken from a point distant from the joint. We never do it until we are forced to do so, because we have been getting grave results. We had one case of knee-joint ankylosis in which we took the interposing flap from the fascia lata over the trochanter major. We took enough to cover all the bone surface, and did not have to use the paraffin. When the time comes that we cannot do it in that way, then the paraffin is in readiness. Dr. Taylor, of Baltimore, has recently been using wax; Baer used hog's bladder, and that is, of course, eventually absorbed, but the idea is to use a material that is not absorbed.

Let the records show that the pick did not penetrate the skull; an area of parietal zone showed great vascularity just beneath the point where the pick struck the skull. There was firm fixation of the pia and dura and a cystic formation over an area the size of a half-dollar. These adhesions were soft and separated easily, and the pia and dura were freed entirely to the midline. A moon-shaped portion of the dura was taken out, and a moon-shaped piece of paraffin was inserted between the dura and pia extending clear to the septum. This was secured in position with catgut passing through the dura. A portion of the moon-shaped flap was removed over the paraffin plate so that the paraffin will come directly in contact with the bone. Then the osteoplastic flap was put back in position and fitted accurately into its place. Through this means we hope to prevent the pia from becoming adherent either to the bone or to the dura at the point of irritation. There is no encapsulated cyst beneath. The soft tissue flap will be sutured with horsehair. The head will be encased in a starch bandage, and the patient will not be permitted to move for five weeks. The case will be treated as a trauma of the skull.

TRIFACIAL NEURALGIA

HISTORY

HOUSEWIFE, aged forty-four. Entered hospital because of severe pain in right side of head and face. Family history and previous personal history negative.

In 1892, twenty years ago, patient says her teeth were badly decayed. By the time she was thirty-seven years old more than half of them had been removed. None had been ulcerated. She does not think teeth of right side were more affected than those on left. She had suffered from toothache occasionally, but never to an excessive degree.

In September, 1905, seven years ago, she was seized suddenly with a lightning-like flash of pain on the right side of the face. It was so severe that she screamed. The pain disappeared just as suddenly as it came. In a few minutes another attack of pain came, but it disappeared just as did the first one. It left no soreness. Another attack of pain came in two days, the next in ten days. During the next two years the pains would come at intervals of days or months, and during the two years following this she was free from pain of any kind. At the end of this period, four years after the first attack, the pain recurred, but the character of the pain changed. Often it would come on gradually, getting worse and worse, until it became unbearable, and the patient would drop to the floor, writhing and screaming in agony. After one or two minutes the attack would pass off and she would resume her work, but feeling exhausted and unstrung.

Eating or talking is especially apt to precipitate attacks. The patient moves lips and jaws very slightly while talking, as though the jaw were ankylosed.

Two and one-half years ago she entered this hospital and was injected on the right side by Dr. Murphy. The patient states

that alcohol was used. For nine months freedom from attacks was absolute, but soon the pains returned and were as bad as ever. She suffered for two weeks and then entered another hospital in this city, where a second nerve injection was made. It had no effect. During the last year and a half she has never been free from attacks of pain for more than a week or a month at a time. The face is reddened on the affected side only during the attack. The right side of the face is hyperesthetic about the middle of cheek, just below the eye, and on the chin it is analgesic. She never has contracted a drug habit. Never has had any trouble with the nasal sinuses, eyes, ears, or throat, nor has she been injured about the head or face in any way. No muscle paralysis or facial distortions are apparent. Examination of heart revealed nothing pathologic. Urine contained a trace of albumin.

Examination by Dr. Murphy: A strip one-half inch in width, extending from angle of mouth upward and backward, is sensitive and causes spasms of facial muscles when irritated by a pinprick. The region supplied by the infra-orbital nerve has not regained its sensation. The region supplied by the submental nerve is devoid of sensation.

COMMENTS AND OPERATION

Let the record show that this is a classic history; that she has had the periods of relief mentioned from the alcohol injections; that the injections ceased to give her relief.

We will make an osteoplastic flap, consisting of scalp and bone, in the temporal region, with the base at the zygoma. We must be careful not to injure the middle meningeal artery. The dura will not be opened, but is very carefully stripped from the middle fossa of the skull until the inferior and superior maxillary divisions of the nerve are found. These are traced backward to the ganglion. A rubber tube constrictor incircles the head just above the ears. This effectively prevents hemorrhage from the scalp.

The danger of injury to the brain is, I believe, lessened by this operation. Here is the middle meningeal artery now.

We are right at the foramen ovale, and both nerve-trunks are plainly in view. We will cut these nerves and then put some

beeswax—a thin plate—over the foramen, so that the stumps of the nerves cannot unite with the distal portion. There is considerable bleeding, so that we must proceed slowly. As I free the nerve I elevate it with a nerve-hook and then sever it with a knife. In that way I am sure that all the fibers of the nerve have been divided. Beeswax is better than paraffin for this work. The wax goes down as a peg into each of these foramina, and then spreads out above the peg so as to cover the entire opening. The brain will come back as soon as I release the pressure on it. I like the Horsley instrument for this purpose. It is a great protection to the brain. It certainly lessens the chances of doing injury with the retractor.

We are now ready to replace the osteoplastic flap. Remember that the dura has not been cut. We simply allow the bone and fascia flap to fall back in place and suture the skin with silkworm-gut and horsehair, without drainage.

These cases can be treated in a variety of ways: First, we have the injection method; second, the division method with the injection; these are extracranial procedures; third, the intracranial types of operation, the division of the roots inside the foramina, and the closing of the foramina with heterogeneous material, such as the rubber tissue of Abbe, the screws of Mayo, and ligatures; the wax plug of Murphy. Next is the removal of the Gasserian ganglion and the removal of the sensory root of the ganglion—Frazier's method.

Osmic acid injection is the plan we usually resort to first, with the division of the nerve at the intracranial foramina. It gives a relief varying from fourteen to eighteen months. Patients who have gone as long as seven years without recurrence are considered cured. A number of them have gone three years, a smaller number four years, but the average case recurs in from fourteen to eighteen months. The second osmic acid injection gives about the same period of relief as the first. The third or fourth injections are not so successful. The alcohol injection gives relief for from six to ten months. Whether the injection is given above the zygomatic arch or below it seems to make but

little difference. To us the injection below the zygomatic arch seems a safer and more certain procedure.

We have done altogether some 16 or 18 Gasserian ganglion removals, that is, complete removals, securing in all cases in which we have been able to get reports permanent relief. It is the desirable operation if it can be executed, but we have found it difficult to execute on account of the bleeding, and therefore we have resorted to the easier and safer procedure of extradural exposure of the inferior and superior maxillary division of the nerve. We have divided them and placed a paraffin or wax plug a little larger and thicker than a nickel over the foramen, and pressed it in and firmly interposed it in that position. The pivots which are made by pressure prevent the material from sliding and those pivots are in the foramen ovale and foramen rotundum. The superior maxillary division is not disturbed in this procedure.

In the removal of the Gasserian ganglion the roots are not divided until the upper layer of the dura is entirely freed from the ganglion; then the roots are divided and the Gasserian ganglion evulsed, holding both of the roots in the same root-retracting forceps, which is of the pattern of the Mayo cystic or common-duct forceps. It makes a splendid evulsor, for the depressed root is removed when it passes through the upper half of the dura on the surface of the ganglion and then backward and upward over the temporal elevation until the posterior root is seen shining in the field. This is hooked up with a blunt hook resembling that used by Alexander in extracting the round ligament, or by the oculists in doing myotomies on the eye muscles. When the root is thoroughly hooked, traction is made on it. It is not cut off, just simply dropped, because it has been detached from its central position; it never reunites because there is a permanent degeneration of the part, that is, the pons having no neurilemma, its axons are permanently incapacitated, as was shown in the experiments of Frazier.

A starch headcap will be put on in this case and not removed for two weeks, when healing will have taken place.

TUMOR OF SPINAL CORD

HISTORY

FEMALE, aged fifty, housewife. Father died of arteriosclerosis at the age of seventy-four. Mother died from nephritis at the age of fifty-nine. No history of malignancy or tuberculosis in family. Menstruation began at fifteen, regular up to thirty-five. At the age of thirty-three patient began passing blood-clots with each menstrual flow. This lasted until she was thirty-five, at which time she had a miscarriage. Following this she was cureted, and since then she has not menstruated. Had one child at twenty-eight, normal labor and puerperium. No laceration.

Had measles, mumps, and whooping-cough when a child. Following her miscarriage she had a severe infection due to the curetage, and was in bed for three weeks. Three months following she had a severe pelvic infection which subsided in a month. There was no operation at this time. Two years following this patient had an attack of bronchitis for three weeks.

Present Trouble.—In February, 1908, she had intermittent attacks of pain in the right elbow and forearm. Patient thought it was due to rheumatism. The attacks came on especially after writing much; were more frequent at night, often causing the patient to awake from sleep. These attacks of pain occurred on an average of one every two or three weeks, without change in character. In December, 1909, patient began to notice a numbness in her right hand, involving the fourth and fifth fingers. This numbness gradually extended up the ulnar border of the arm.

In January, 1910, she was examined by a doctor and the sensation in the right arm was more pronounced on the ulnar border, extending to the shoulder, also in the right leg extending

from the toes up to the waist-line, and in the left leg from the waist-line to the knee. Since this time the numbness has gradually increased until sensation is entirely absent from the toes to the waist-line, and there is partial anesthesia from the waist-line up to about the third rib, and in the arm and fingers to the shoulders; more pronounced on the ulnar side. The first disturbance was noticed December, 1909, when, on attempting to step into a carriage, she noticed a heaviness in the right leg. This gradually increased in degree until now she cannot use either leg. Since the patient began to notice the trouble she has not had any difficulty with bowels or bladder. There is no disturbance in eyes or ears. The patient was operated on at that time, but in February, 1912, the symptoms recurred.

COMMENTS AND OPERATION

Yesterday we operated on this patient for a tumor of the spinal cord which was situated between the spinous processes of the seventh and the sixth cervical vertebræ. It was a recurrent case. The patient originally came to Dr. Hugh T. Patrick, who examined her, and he was unable to make a diagnosis of tumor at that time. Some three months later a diagnosis of tumor was made. Then she went along for six or seven months before she was operated on. The symptoms she had originally were pain in the right and left arms, then pain in the limbs, followed by hemiplegia and then a paraplegia, so that she was practically paralyzed, except for a few muscles in her upper extremity. The tumor was situated between the sixth and seventh spinous processes of the cervical vertebræ. At the first operation it was situated between the fifth and sixth, shutting off the nerve-supply to the brachial plexus, except the two upper roots.

The trouble was diagnosed as a tumor on the posterior surface of the cord. We cut down on it at that time,—about two years ago,—and removed a tumor that came out in the posterior fissure of the cord. It affected sensation first and then motion. The tumor was about an inch and a half long, and in diameter about the size of my little finger. We lifted it out without traumatizing

the cord, and the woman made a complete and perfect recovery. She resumed her usual work; in fact, she was well part of last winter, so that she did not have a maid and tended the furnace. This shows you how fully restored her function was. Then, about the middle of January, 1912, she began to have discomfort in her arm, the pain began to increase in severity, extended higher up, until finally she got ataxia of gait. Dr. Patrick concluded that she had another tumor, and that it was lower down than the first—situated between the sixth and seventh spinous processes of the cervical vertebræ and on the right side.

We operated on her yesterday, reopened the spinal cord, freed it from cicatricial tissue, and took out a second tumor, about half the size of the original one. We closed the cord membranes carefully with fine catgut sutures, then put in deep and superficial catgut sutures and coaptated the skin with horsehair. A collodion gauze dressing sealed the wound. It was situated near the posterior root of the spinal nerve and pressed on the anterior and anterolateral columns. After it began to grow the motor symptoms were more pronounced on the right side. The operation was done without any untoward symptoms. Last night she felt better than she had for months; she did not have any pressure pain in her cord. She will undoubtedly go on to recovery. The tumor proved to be of the endothelial type, originating from the lining of a lymph or blood-vessel. These tumors are really of the nature of sarcomata. Whether this tumor will recur or not we do not know, but I should say it probably will; but if recurrence does not take place in two years, or in a year and a half, we shall feel that we have been well rewarded for the effort. It is a better reward than one usually gets.

[When the patient left the hospital, about three weeks after the operation, the wound had healed per primam and she was apparently entirely well and free from pain.—Ed.]

CHRONIC MASTITIS

HISTORY

HOUSEWIFE, thirty-two years old. Entered Mercy Hospital because of a tumor-mass in the left breast. She has been married fourteen years and has borne four children. She has had two miscarriages at about the third month each, following overwork. She always has nursed her children. Past life has been free from all sicknesses of a serious nature. A cousin died of carcinoma of the breast.

Ten years ago (1902), while nursing her second child, she had an excessive amount of milk in both breasts. She wanted to check this excessive secretion, and for this and no other reason, she is sure, she put the baby to the right breast always. She states that within four weeks the left breast became somewhat hard and slightly tender. This tenderness persisted and increased in severity, but she thought that the breast had "caked" because she did not put the baby to it, and therefore paid little attention to the tenderness. Four months after the baby was born the breast had become so sore that on the advice of a neighbor she applied a clay pipe to the nipple and sucked out several tablespoonfuls of a material that she feels sure was thick pus. It had a sour, milky odor. The breast (left) remained tender and firm beneath the nipple and to its outer edge. This has remained so ever since.

One and one-half years later she became pregnant for the third time. The breast became slightly more tender, but no milk flowed, nor did it discharge again. Her fourth child was born within two years, and the breast was the same as before, with the exception that the mass was a little harder and a little larger. At no time since its first appearance has the tenderness and firmness disappeared.

In the fall of 1909, while in a crowded street, she was struck a sharp blow on the breast by a person's elbow. For one-half hour she suffered considerable pain. She cannot remember that the breast was more tender than usual during the following day or two. She thinks that the mass has increased in size and firmness very gradually, but that it is less tender now than during the years when she was having children. The skin never was discolored or inflamed at any time. The tumor was never nodular or rough. The axillary glands cannot be palpated. Four months ago (December, 1911) she first noticed that the nipple seemed to be held firmly to the hard mass beneath it. The mass is now of about the size of a lemon, is very firm, and seems to be fixed in the breast substance as well as to the nipple and the skin about it. Heart, normal. Urine, negative.

COMMENTS AND OPERATION

Dr. Murphy: As I remember, there was a yellowish discharge from the nipple. That should be put in the history, if it is not there. You cannot interpret that as pus, because then you have a thick, yellowish discharge from the lacteal glands. It is not at all uncommon. This history is exceptional on account of the period of time that elapsed between the trauma and the manifestation of the tumor. It is three years since she had an injury. You will note again that she had only one injury, not many injuries; that following this one injury she had considerable pain for half an hour, then it subsided, and the whole thing disappeared. When was the discharge from the breast?

The Intern: Three years ago.

Dr. Murphy: Before the injury or after the injury?

The Intern: A year before the injury.

Dr. Murphy: She never had a suppurating breast. She nursed all her children preceding that time, but not the last one, and with the last child she had no mastitis and no inflammation. The breast was a little tender, but the pain was not severe. There was no suppuration. A year after that time she received

a trauma, and two years after that time she comes here with a lesion in her breast.

The question arises, has this woman, as the result of the trauma, a carcinoma? Has she a sarcoma, or has she an interstitial mastitis? What is her temperature?

THE INTERN: The temperature is normal.

DR. MURPHY: There is evidence of acute infection. We examined this woman's breast yesterday and we found that she has a diffuse infiltration of the breast. When the nipple is retracted, the skin over it is fixed. She is only thirty-two years of age, stout, ruddy, and robust. That means what? That means, if we interpret this as a sarcoma or carcinoma, that we have to deal with a condition that is ultimately going to terminate in a cancer death. Then, were this woman sixty-two years of age, she would have a much better life expectancy than she would have at thirty-two. When a woman of thirty-two comes to you and is stout, with a lesion in her breast that has existed for a considerable period, practically in 100 per cent. of the cases she will have a cancer death. That is an admission, but it is a fact. When you find a tumor in such an individual, you base the life expectancy on the individual rather than on the statistics. Women who are stout, who have a large amount of fat, particularly when they are young, have rapid transmission of the malignant epithelial cells through the lymphatics. On the other hand, if they are thin and aged, the lymphatic circulation is slower; the new connective-tissue formation encapsulates and retains the epithelial cells *in loco* a much longer period of time. That is what one must assume in making a prognosis in such a case as this.

There is here the typically retracted nipple. There is a mass under it. It has a sense of fluctuation. There is no evidence of involvement of the axillary glands as yet. We are going to take out this tumor as it is, then bisect it, examine it, and determine what we will do with the axillary glands. This mass must come out, because whether it is malignant or not, there is only one treatment, and that is its removal and its complete removal at this time. We will have skin tissue in great abundance.

In making the skin incision in this case we are not going into the axilla, but above it. It will encircle the breast. The incision will extend down to the pectoral fascia. I will not take out the pectoral muscle at this time, but will remove the aponeurosis of the pectoralis major muscle. In doing these operations we take into consideration the line of lymphatics leading from the tumor and the position which the tumor occupies in the breast. The incision curves above the muscle, so as not to have the scar line crossing the axilla. The fascia between the pectoralis major and pectoralis minor should always be removed because it contains lymph-nodes which may be the seat of metastases now or become such later. The next thing is to take off this breast and make an examination of it before we attack the axilla. This is done by freeing the breast from the chest-wall, removing all the fascia containing lymph-vessels and lymph-nodes. We will now incise the mass. It contains a purulent fluid. It is not necessarily pus. It is questionable in my mind whether that tumor is malignant. This breast is riddled with infiltration and degeneration, and it contains either pus or it is a retention of the secretion from the epithelial cells lining the lactiferous glands and ducts. We will not take out the axillary lymph-nodes at this sitting. The multiple "infections" here are accumulations which are probably retention cysts with mixed infection. We will have this mass examined microscopically, and then, if necessary, if it shows signs of malignancy we will take out the axillary glands later.

This woman is very stout, and the removal of fat from her axilla and the removal of the lymphatics of the axilla will give much greater edema and swelling of the arm even with the implantation of the pectoralis major than would occur in the average case. That is why we do not wish to go into the axilla unless this tumor of the breast is malignant. If this woman should get lymph stasis, stout as she is, her arm will become practically useless. We have not disturbed the fatty tissues about the vessels at all. She will have no lymph or venous stasis from anything we have done. These axillary glands will be removed in two weeks from now if the tumor proves to be

malignant. There is apparently no involvement of the lymphatics. In this case infection would be just as likely to cause enlargement of the glands as would metastasis from malignant disease. We will close the wound in the usual manner with silkworm-gut. A gutta-percha drain is inserted to permit the wound secretion to escape. It is taken out after seventy-two hours. There is no tension on the line of sutures, and still the skin is in much greater danger of necrosis than the average skin-flap, because there is so much fat here.

In these infections of the breast there is the same tendency to glandular involvement as in carcinoma. This is not the usual type of pus infection. When the bacteriologist makes a smear and culture from the breast, it will probably be negative, because the fluid in the breast is the lacteal fluid usually found in the chronically inflamed and irritated breast. The cicatricial tissue has obliterated the outlet of the lactiferous ducts, and therefore we have in these cases a lactiferous fluid—merely a fatty, creamy fluid, in place of pus.

That was one of the greatest evidences against this being a malignant lesion. Primarily it was not a malignant lesion. We are certain of that, because there were multiple foci and they were a considerable distance apart. That speaks against malignancy at once, and that formulates my opinion as to this being a chronic mastitis more than the mere presence of the fluid, because not infrequently you find that type of fluid present when there is a malignant lesion, and particularly a rapidly growing malignant lesion, and the younger the individual, the more likely is that to be the case. These lactiferous gland cells are active at this time of life, and, therefore, fatty degeneration and the secretion of milk are going on at the same time.

The additional danger of removing the tissues in the axilla is comparatively small, taken as a whole. The percentage of danger in the individual case following the removal of the axillary zone is over 80 per cent., the risk of amputation 0.8 per cent., the mere removal of the breast is practically no risk at all, the removal of the axillary glands with the breast has a small percentage of risk.

All the tension sutures which we have put in are silkworm-gut. The skin edges are coaptated with horsehair. We use horsehair in the skin because it is elastic; silkworm-gut is inelastic. Silkworm-gut cuts the skin; horsehair does not, Horsehair does not cut the skin because it stretches, but it will stretch only once. It does not rebound. If I stretch it again, it breaks. That is why it is ideal suture material for the skin. It is elastic, but it does not again contract; it gives without contracting. Silkworm-gut does not give any more than wire does. It produces skin necrosis, and when that occurs, you are very likely to get infection with saprophytes, so that the preservation of the margin of the skin is very important. In these cases we have to make such long flaps, and we have to remove the fascia and fat from beneath the skin, so that it has not been uncommon to have marginal necrosis, and it is not uncommon to get saprophytic infection after necrosis has taken place.

In these cases, if you do not provide for drainage of the secretions, which sometimes are considerable in amount, especially when there is much adipose tissue, as in this case, they accumulate and interfere with healing. Before agglutination of the surfaces occurs, before definitive adhesions can form, it is essential to provide for the removal of all of that secretion. Absorption in a tissue of that kind is extremely slow, therefore you hasten the consolidation of the scar by putting in a drainage-tube for forty-eight or seventy-two hours, thus allowing the wound secretion to escape. Then take out the drain. If you keep the drain in longer than seventy-two hours you are likely to get saprophytic infection, and then you will have secretion pouring out for twelve or fifteen days. You can open a joint, and if you will close it again, there will be no more tendency to ankylosis than if you had not touched it. If you put in a drain for seventy-two hours, or a week, or ten days, you have ankylosis in over 90 per cent. of the cases. If you follow the lines of these principles in surgery, you will find the same thread running through every strand, figuratively speaking. For a time we put in flap drains; that is to say, we used gutta-percha tissue without a wick. The wick acts as a siphon for

a few hours only. After that time it acts as a plug. Rubber tissue around the wick makes drainage around the wick but not through it. The center of the drain is plugged. A strip of gutta-percha allows the fluid to escape when it gets under considerable tension. We want it to escape onto the dressing as it is formed, and, therefore, a large dressing is applied and fairly firm compression is put on the whole dressing, so as to keep the wound surfaces approximated as well as we can during the early stage after the operation. That approximation tends to lessen the hemorrhage. The approximation of muscle tends more to the lessening of hemorrhage probably than any other tissue, except when you come to renal tissue or adrenal tissue. This is beautifully illustrated by Sir Victor Horsley in his management of the oozing from the surface of the brain. He takes out a piece of the temporal muscle and applies it to the oozing area, and there is something coming from that muscle that acts as a hemostat. Just what it is has not yet been definitely settled. In operating on kidney tissue we find that hemostasis takes place quite rapidly. In renal surgery always open the kidney from the pelvic side. In going through the cortex you immediately have a great gush of blood and you think the patient is going to bleed to death in a few minutes, but just hold the kidney back and put on hand compression for two or three minutes, open the kidney, and there are not more than two or three vessels bleeding. The reason for that is the action of the renal cells. There is really no very great difference between renal and adrenal tissue, so far as the hemostatic effect of local compression is concerned. In making the approximation of the wound surfaces it is important to get accurate approximation, so as not to have primary effusion. The more tension you have the more likely there is to be oozing, yet the packing stops it.

As the axilla was not excavated in this case, there is no danger of a sponge being left in there. When the axilla is excavated, a sponge count is made, as in a laparotomy, because sponges have been left in the axilla a number of times; they have been left in the triangles of the neck, so that whenever we make a complete breast operation we always keep a record of the sponges

just the same as in our laparotomies. This was merely a surface amputation, hence there was no need of keeping a count of sponges.

Let the record show that there was no count kept of the sponges; that the axilla was not cleansed. Let the record show further that there were numerous foci of cicatricial tissue an inch and a half apart, extending through the upper and outer half of the breast; that in these separate foci were cavities filled with a creamy, yellow, purulent-looking material; that around these cavities we found considerable infiltration of tissue; that in no place in the breast did we find definite, hard fibrous masses or a mass without the infected focus, resembling the macroscopic picture of a carcinoma. From the fact that there were multiple foci; from the fact that this patient is thirty-two years of age; and from the fact that there was no dry focus of infiltration, we believe that this is a mastitis with retention and possibly with infection. [This diagnosis was later confirmed by the microscopic examination. The wound healed per primam, and the patient made an uneventful recovery, leaving the hospital in perfect health three weeks after the operation.—Ed.]

RECURRENT OVARIAN CYSTOSARCOMA

HISTORY

FEMALE, aged forty-six. Family and personal history negative. Menopause four years ago; has not flowed since.

Present Trouble.—Began four years ago (1908). Patient noticed that her clothes did not fit. Then noticed a distinct swelling of abdomen, more marked on left side, but gradually becoming general. This swelling continued to increase slowly in size, without pain or other discomfort, until about three months ago. At that time she noticed a hard mass about the size of her palm in the left lower quadrant of the abdomen, seemingly just inside the abdominal wall. It appeared to be fixed. It was not painful or tender, but increased rapidly in size. The general abdominal swelling also increased more rapidly, and patient began to have difficulty in breathing. Bowels have been becoming more and more constipated. Urine has always passed freely and nothing has been noticed in it. Patient lost ten pounds in weight in three months. Is growing steadily weaker. Has had no chill, night-sweats, or fever.

She was operated on in May, 1909, an ovarian tumor being removed, and after that felt well until the last Thursday in November, 1910. She was a pall-bearer at a funeral on a rainy day, and just during monthly period, which usually lasted three days. The flow stopped after the first day, and for the next two months she did not flow. Patient was up and around a week. Flowed February and March, 1910; flow was very slight in amount and lasted only one day. Since then she has not flowed at all. Has not felt entirely well since. Has a feeling of rawness internally over whole abdomen. Since operation patient has been feeling about abdomen for a possible return of the tumor, and the last day of June, 1911, she felt a tumor about the

size of a dollar in the right lower quadrant of the abdomen. It was very hard, not painful, and would slip away from under her fingers. This has increased rapidly in size and has the same consistence as at onset, but has grown to such an extent that it entirely fills the abdomen and resembles a pregnancy. Since last operation patient has lost twenty-five pounds in weight and has also lost her appetite. The tumor was again removed. Says it was an ovarian tumor. The patient made an uneventful recovery. Bowels regular. No urinary symptoms.

May 7, 1912, patient returned to the hospital. Since her last operation, in August, 1911, patient has felt well. When she left the hospital she had been instructed to return every three months for reëxamination. May 3, 1912, patient was examined by Dr. Murphy and told to return for operation. She has had no subjective symptoms.

COMMENTS AND OPERATION

Dr. Murphy: What date was the first history written?

The Intern: 1908, when patient was forty-two years of age.

Dr. Murphy: This was at the first operation. How about the second operation?

The Intern: The second operation was done one year later.

Dr. Murphy: What I wish to know is about the pathologic findings in the first operation.

The Intern: It does not state in the report.

Dr. Murphy: The history of the pathologic findings should be there. I remember it very distinctly. We thought this an adherent cyst, but the pathologist said it was a cystosarcoma. The subsequent history shows where the pathologist helped us out at this time; it shows why this patient is having a recurrence. It was a mixed-cell sarcoma of the cystic variety. This time the tumor is much smaller than at any previous time. We hope that there are no adhesions to the intestine. She came in without any symptoms at all; came in on instructions to have repeated examinations made so as to be on guard before the tumor attained any great size. We will open the abdomen in the midline, between the umbilicus and the pubes. We are going through

cicatricial tissue here, and are going through it with caution. On going through the peritoneum and entering the abdominal cavity, we find that there are adhesions present. They are the soft, friable adhesions one commonly sees in tuberculosis—the so-called "cobweb adhesions." There is a mass here that looks like an infected gland, but as there are no glands in that position, it is probably a secondary neoplasm. The bowel is fixed to the anterior abdominal wall. These are more of the cobweb type adhesions and are easily freed.

The ovarian cyst is now plainly visible. It is of a dark color. The patient is having metastases, and is in a more serious condition than she was in before. There is a neoplasm on the wall of the small bowel of quite a size. It is a secondary neoplasm. We must ascertain whether there are many of these before we attempt to do anything. There is also a larger cyst here. It appears to come well up into view, and is attached away over on the left side. There is another implant in that region, also of the sarcomatous type. The neck of the cyst is adherent to the anterior abdominal wall. It is going to be difficult to free this. The attachment to the anterior abdominal wall is probably the place from which this cyst springs—from the peritoneum.

I will try to remove the base of this tumor with its peritoneum. I am now examining the intestine to see what relation the colon bears to this tumor. The colon is free from neoplasm. When we get the line of demarcation between tumor and normal tissue established, we should be able to enucleate this with comparatively little trouble, and we should get into what is known as a bloodless zone.

I want to call attention to the physical characteristic of this neoplasm, namely, that it tends to maintain its cystic character all the time. It has not gone through the peritoneum. As far as we can determine now, it is just adherent to the serosa. We can easily remove the tumor. Just shell it out. On that you can base a positive diagnosis alone. You do not need to have a microscopic examination made to tell the story. Then put a puckering string of catgut around this abraded spot and turn it in.

The uterus is about normal in size. Now we must cover as

much as we can this abraded surface of the peritoneum with the normal peritoneum of the opposite side. It is going to be a little difficult, still I think I can accomplish it because this woman is so very thin. We are able to take up the peritoneum and approximate it across the line and cover the abraded area fairly well, notwithstanding the fact that the abrasion of the parietal peritoneum was of considerable extent. However, in a stouter woman we would not have been able to do this.

We will close the peritoneum with a continuous catgut suture, and approximate the deeper structures by means of a figure-of-8 silkworm-gut suture, putting in three or four to insure a perfect result. The skin wound is closed with a continuous horsehair suture, dusted with bismuth subiodid powder, and sealed with collodion gauze. Over this is placed the usual dressing, held in place with the abdominal binder.

We were able to cover this raw surface better than I thought we would. The abraded area is fairly well protected with peritoneum on the inside, and there remains only a small zone for adhesions. This tumor will in all probability recur and go on to a fatal termination, possibly with greater rapidity than it did before the last operation, because it has attacked a new zone—the peritoneal surface.

This patient will be put on large doses of arsenic, hypodermically; that occasionally retards growths of this type, and occasionally retards them in a very striking manner. I will not say that I have seen a cure result, but I have seen large tumors disappear and then I have seen them reappear, but it gives the patient relief from pain because it stops the pressure of the growth.

The history of this case is the history of many of this class of cases, with one exception—the prolongation of life is away beyond the average period. We have succeeded in saving her life for a much greater period than is possible on the average. This cyst was not like any of the other cysts that one sees. If you take a section of this cyst-wall, you will find that it is not lined with epithelial cells, as is the case in all ordinary ovarian cysts and as you see it in sarcomatous conditions of the ovary.

This is a malignant hyperplasia of the connective-tissue elements, of the mesodermic element, and not of the ectoderm or endoderm cells, and it is for that reason that the tumor continues to recur and recur, but it does not recur in the gland itself. The recurrences were surface recurrences. The same thing occurs with implants due to ruptured ovarian sarcomata or ruptured papillomatous cysts. Papillomatous cysts cause the least numerous deposits of the peritoneal surface. When papillomatous cysts rupture, the peritoneum becomes studded all over with small tumors. In the last three months we have had three cases of this variety. In two of them we made some effort to remove the tumors. The case in which we made no effort to remove the growths has gone on to a fatal termination. In the two cases in which we made a partial effort at removal, notwithstanding the fact that the masses were not all removed, the effusions were relieved by thyroid and arsenic treatment. We gave large doses of arsenic, two and three grains at a time. Sodium cacodylate is the form of arsenic best tolerated by this class of patients. A few weeks ago we received a letter from a patient who some three years ago had had the same type of papillomatous cyst that could not be operated on. Everything was studded with tumors, but all the evidences of the disease have disappeared under the arsenic treatment.

We cannot say whether these tumors are malignant or non-malignant. We know that papillomata disappear on the surface of the skin (warts), a whole crop appearing and disappearing without any known cause. The same thing occurs in connection with papillomatous cysts in the peritoneal cavity. The microscopist reports all of them as being malignant. You can see why. Because the epithelial cells have passed beneath the basement-membrane. Clinically, we do not regard them as being carcinomata, and the fact that they disappear makes us want to include them in the non-malignant class of tumor. This, however, is not of that variety at all; it is a connective-tissue cyst formation, not an epithelial cyst formation. We know very little of this variety of sarcoma. We recognize its cells; we recognize its tendency to malignancy; we also know that it will

appear in one ovary and after some months appear in the other ovary. We also know that it is the tendency of sarcoma of the ovary to become cystic. We do not know, however, why this metastasis occurs in the other ovary. We know that in sarcoma of one testicle the rule is for the other testicle to become involved, even when the one first involved is removed. If a patient has a sarcoma in one kidney, he always gets a second tumor in the other kidney. That is in refutation of the belief that these tumors are parasitic in origin. Why should these tumors come in both glands in just that way if they are parasitic in origin? The same is true of the infective variety of lesion. We know that when a patient has tuberculosis of one epididymis or of one testicle, he is in all probability going to get tuberculosis of the other epididymis or testicle, not as a primary lesion but always as a secondary lesion, showing that you can exclude these lesions from being parasitic or bacterial in origin.

RETROVERSION OF UTERUS

HISTORY

Female, aged thirty-nine, married, has three healthy children. Was admitted to hospital because of lumbar and pelvic pains and extremely painful menstruation. Pregnant five times, the second and fourth pregnancy resulted in miscarriage at about the third month. The other pregnancies went to full term with normal deliveries.

In December, 1908, had an operation for acute left-sided pelvic pain, with vomiting, but no chills or fever. Previous to this, patient had not missed a menstrual period, but on close questioning she stated that the amount of flow and duration of the two periods preceding the operation were very much diminished. Two days preceding the operation she developed a sudden severe uterine hemorrhage, accompanied by pain and vomiting. This was independent of the menstrual flow. The abdomen was opened, but patient can throw no light on the pathology found or the nature of the operation performed. Since the birth of her first baby she has been troubled with severe lumbar pain, which obliges her to be in the reclining position most of the day. She has pain in the pelvis. There is no variation in the amount or duration of her menses in that time. She has no urinary symptoms.

COMMENTS AND OPERATION

Dr. Murphy: How old is she?

The Intern: Thirty-nine years.

Dr. Murphy: Her menstruation all her life, as you will note, has been painful and of the obstructive variety. Then she came to the period when she had to have an operation, the nature of which I do not know, by which the uterus was sup-

posed to have been restored to position, but her condition has not been materially changed. She is still having her painful menstruation, and she has now a retroversion of the uterus of the third degree—the superlative degree. The uterus is resting in Douglas' pouch. She has some obstructive symptoms, and for that reason we are going to dilate the cervix and do a Pozzi operation on the cervix.

You will recall a paper read by Pozzi in this country in 1904, when he outlined the plan and reason for his operation for obstructive dysmenorrhea. Obstructive dysmenorrhea of the type mentioned is thought to be due to retention in the external os, and it is due to stenosis, first, of the external os; second, to dilatation of the cervix between the os externum and os internum. We have since that time had quite an experience with this operation, and it is one of the most gratifying operations we have, with little hazard and little inconvenience to the patient. It is so easily done. It consists, in the main, of producing a permanent dilatation of the os externum—the mere dilatation of the cervix does nothing. It contracts again and always seems to become smaller after dilatation than before.

You will remember in the early days of the American operation, so called, for hemorrhoids, we overdistended the rectum and the patients frequently got incontinence of feces. It is entirely different in the uterus. The uterus is a musculofibrous organ, and the fibrous tissue has a tendency to contract after it has been traumatized.

The patient is placed in position, the anterior lip of the cervix is grasped with a vulsellum, and the uterus drawn to the vulva. We will first dilate the cervix with forceps dilator. I have directed that dilator upward because the uterus is retroverted to the superlative degree. This is a temporary dilatation and is done with moderate force, as I am going to make a division of the cervix and do a typical cervical repair. Having secured full dilatation of the cervix with the various dilators which are usually used, we will proceed to curet the uterus. This curet is a dull curet, the kind we always use. Now you can get an idea of the size of this cervical canal. The cervix permits

my finger to enter clear back to the first joint. It shows that this was a potent element in the production of the pain. It was an obstructive dysmenorrhea from an irritation of the os externum. Pozzi's suggestion for treatment consists in taking a wedge-shaped piece out of the muscularis of the cervix. The purpose of taking that out is to permit the approximation of the squamous epithelium on the outside of the cervix to the columnar epithelium on the inside, and thus produce a contraction of the cervical canal. When the two surfaces are united transversely with catgut you can never have re-formation of the divided portion of the cervix, and that insures a patent os externum. The os externum is always larger than normal. The operation is a simple one, easily done, and always gives relief. Two stitches are put in above and two below. This is just what we would do in a case of web-finger. If you leave the divided septum between the two fingers without suturing, the patient will come back with the web-finger returned as bad as before, if not worse; but if you connect the epithelium-lined surfaces in front and behind the web, there is no subsequent contraction. Two stitches are usually sufficient. That insures a permanent patency of the os externum.

To recapitulate: First I took out a V-shaped piece of muscularis from the cervix. That permits of approximation of the outer and inner surfaces of the mucosa, the squamous cells of the vaginal portion of the cervix, and the columnar cells of the endocervical mucosa. The stitches are put in so as to catch the columnar epithelial cells on the inner side, and the squamous epithelial cells on the outer side, bringing those into accurate apposition, so that there will be no possibility of a transverse healing. That is, the transverse incision is sutured longitudinally. Restoring that uterus to its normal elevation in the pelvis would not relieve this patient of her dysmenorrhea as long as this os externum remained closed as it was. It would simply relieve the pain in the back after menstruation, but would not relieve the first day's pain during menstruation. Ordinarily, however, this is the only operation that these patients need to have to get relief. The uterus is allowed to recede, and the vagina is packed with sterile gauze strips.

In the evolution of the treatment of retroversion we have had many discussions and many differences of opinion, and the result has been that we have had many operations devised and performed. How is a retroversion evolved? How does a retroversion occur? In a retroversion of the uterus, occurring either suddenly or gradually, it always occurs with an inflammatory condition in the pelvis which finally draws the uterus into Douglas' pouch and fixes it there, though in the enormous percentage of cases it occurs without fixation or adhesion in Douglas' pouch. The weight of the uterus and the pelvic pressure are all carried by the cervicocorporeal portion of the broad ligament. This is what keeps the uterus from descending. Intra-abdominal pressure on the uterus in the normal position is brought to bear on the posterior surface of the uterus, pressing it forward toward the bladder. As the retroversion occurs the uterus tilts backward more and more, and the round ligament becomes longer and longer, until finally there occurs the superlative degree of retroversion. The intra-abdominal pressure is now on the anterior surface of the uterus and the round ligament—that is, the balancing rope or cord of the uterus—passes in front of the cervix, but if it is torn from its attachment and passes behind the line of the cervicocorporeal attachment of the broad ligament, it is a restraining ligament rather than a supporting ligament, and it holds the uterus backward in place of holding it forward.

In the treatment of retroversion you have to reëstablish the anterior supporting ligament, which is what? The round ligament. Unless that can be reëstablished in its normal relation one has a failure in results. Wherever one attempts to do this with adhesions in the pelvis, nothing but failure can result. It has then been attempted, under unfavorable conditions and under conditions which our knowledge of mechanics as well as of pathology did not justify; that is why the Alexander operation has gradually come into disrepute.

For a number of years we did the ventral fixation operation for retroversion until we learned that that was not the best procedure for retroversion: that it is mechanically incorrect. We will now place the patient in the dorsal recumbent position

and open the abdomen so as to correct the position of the uterus. The incision is made in the median line, between umbilicus and pubes. I do not know what adhesions exist here from the previous operation. Something appears to be adherent beneath the peritoneum, so I will open that with greater caution. When incising the peritoneum, never cut between the two forceps that grasp it, but always cut on the slant, or incline. The air rushes into the abdominal cavity, and that protects one against accident or division of the omentum or of the intestine. By cutting between the forceps we may produce serious trouble. The wound edges are retracted so as to give us better access to the uterus.

I will now endeavor to get the fundus of the uterus up out of the pelvis. I can feel the cervix pressing through the anterior vaginal wall, and back of that I feel a little fibroma which I did not recognize in the original examination. I am trying to catch the broad ligament. I have succeeded in getting my finger under the broad ligament. I have the heavy fundus that was down in the culdesac. There is also a fibroma in this fundus. There is a fibroma which I thought was in front of the cervix. I have pulled free the adhesion that existed. This patient is a nullipara, and I am not at liberty to take out the uterus, but I will enucleate the fibromata, which is the only thing I am justified in doing. This accentuates, more than the ordinary case, the significance of the downward displacement of the round ligament. The round ligament is down almost to the cervix.

Now, doctor, I want you to read again about the duration of her menstruation.

[The intern reads history.]

This will be just an enucleation—a myomectomy. I will cut through the capsule of the fibroma and enucleate it. These fibroids have in no way interfered with her menstruation. In fact, it is the opposite. In place of menstruation increasing in frequency and increasing in quantity, the menstruation has diminished in quantity and diminished in frequency—is that right?

THE INTERN: Diminished in frequency, but not in quantity, doctor.

DR. MURPHY: Diminished in frequency, but not in quantity? Thank you, doctor. You will remember, in the many discussions we have had here concerning fibroma, that one of the things that we have endeavored to accentuate has been that menstruation is influenced by fibroids in proportion to the relation of the fibroid to the mucosa. These fibroids are subserous, and subserous fibroids do not influence menstruation at all. These are striking examples of that situation as far as these lower fibroids are concerned.

We will make an entropium of this serosa, so as to avoid having any abraded or raw surface, which might lead to the formation of adhesions. In the hemorrhage from fibroma you have to take into consideration not so much the size of the fibroma as the relation of the fibroma to the mucosa. A fibroma not larger than a filbert situated in a submucous position—a submucous polyp, if you please—influences menstruation more than a fibroma six inches in diameter situated subserously.

There is still another fibroma below. Let us see what relation that bears to the trouble. It appears to be deep in the muscularis. There is practically no bleeding at all from that as I enucleate it. I am going to turn in the cut edges of this serosa as I did in the case of the other tumor. Catgut is used for this purpose.

The next step in the operation will be to throw this uterus forward. You can see why any external operation would have been a failure here. The question arises whether this fibroid was present at the previous operation. There is no record of it and no mention of it by the patient. She did not mention it to me and she did not mention it to the doctor who took the history.

I do not wish to leave a raw surface exposed on the fundus of this uterus; if we do, we will have an adhesion to it and that is not desirable at all.

This woman is still in the child-bearing period. We will take off this vulsellum which has held up the uterus and let the uterus come around to its normal position and close up the wound, from which we have a little oozing as a result of the attachment of the forceps.

The next step is fixing that uterus so it will stay forward. Let us see where these round ligaments are. They are far below the dome of the fundus. We will bring those round ligaments well over so as to tack them with catgut sutures to the highest portion of the dome and behind. I throw the uterus forward toward the bladder, where it belongs, and I will fix the round ligaments behind, on the posterior surface of the uterus, so that they will hold it in proper place. Then, you see, each round ligament passes directly upward and outward to the internal opening of the inguinal ring. I am not disturbing the relationship of the tube at all. This patient can become pregnant and that round ligament will elongate, as it should. The round ligament carries none of the weight of the body of the uterus. It merely balances it. The intra-abdominal pressure will hold it in place. I will have to shorten it about an inch and a half. I want to call your attention to the descensus of attachment which has taken place, which is a significant thing. The normal descensus takes place when this patient is changed to the sitting position. She has this rope or ligament fixed to the highest point up above the cornu, and that gives the greatest possible leverage advantage one can get from the muscle from this side. Each round ligament is looped behind the uterus, tacked to the fundus with a few catgut sutures, and then two sutures unite the loops to each other. That is ample to support the uterus subsequently.

Now comes the closure of the abdomen. I make an ectropium of the cut edges of the peritoneum by turning them out. I wish to prevent the cut edges of this peritoneum from being exposed on the inner side, because if they are exposed we will have adhesions within at the line of division. We have done that now for ten years as a routine procedure. We have had many opportunities to reëxamine these patients and we have been impressed by the absence of organic adhesions at the line of union, compared with what we had before we made the ectropium. We believe it is a very material aid. There is no abraded surface there to which adhesions can take place. So important is that in the avoidance of traction following opera-

tions that we have come to consider that advancement in the next ten years must be on the line of avoidance of organic adhesions of the omentum and intestines to the anterior abdominal wall. The intestinal adhesion that occurs is the most serious one, because you have the intestine attached behind by its mesentery to the spinal column from the second lumbar. We close the peritoneum with a continuous catgut suture.

The next step is the accurate closure of the aponeurosis of the rectus. We will not include in the suture the muscles nor the tissues external to the aponeurosis, because we have learned, from our knowledge of histology, that if you let some muscle or fatty tissue come in between the fibers of the aponeurosis, you have failure of edge-to-edge union and a tendency to spread, with the subsequent development of hernia. The rectus gives support in a perpendicular direction and little support in a transverse direction, so it is important to get accurate apposition of the aponeurosis of the rectus because that is the great transverse support against the formation of hernia. We again use catgut. The skin and fascia are approximated with figure-of-8 silkworm-gut sutures. The skin edges are carefully coaptated and united with horsehair.

Take the history of the case—obstructive dysmenorrhea, pain in the back, discomfort in the pelvis, without any material increase in the quantity of the flow. After the abdomen was opened we found a superlative degree of retroversion. Study of the case further showed that, as that retroversion evolved, the round ligament slid further and further down in its attachment. Shortening of that round ligament by the Alexander operation would not have done this patient one particle of good.

In the cure of retroversion there are two plans of procedure. If you have a retroversion with descensus of the uterus, that is, with prolapsus of the first or second degree, then you have to do some operation which either fixes the uterus to the anterior abdominal wall or you have to take out the uterus. When you take out the uterus and do a complete hysterectomy and shorten the transverse ligament, in a considerable percentage of cases

you effect a cure. When you take out the uterus, however, in other cases, you have finally prolapsus of the vaginal wall and prolapsus of the rectum and prolapsus of the bladder, and you have nothing left to which to anchor these structures subsequently. In an ordinary retroversion in a nullipara without enlargement of the uterus and without pelvic inflammation the Alexander operation is ideal. The operation as we have done it here for eight years is folding the round ligament over the dome of the uterus, suturing it there. This shortens the round ligament and tips the uterus forward.

The wound will now be dusted with bismuth subiodid and sealed with collodion gauze. The stitches will be taken out in about ten days. A large abdominal pad and binder make all snug.

RECTOCELE AND PERINEAL LACERATION

HISTORY

HOUSEWIFE, forty-eight years old, entered hospital for the repair of a rectocele and lacerated perineum. She was married at the age of nineteen and has given birth to six children. She has had three miscarriages. Aside from trouble connected with childbirth she has been free from illnesses of a serious nature.

After the birth of her first child, twenty-nine years ago, her perineum was torn badly. One year later she had a miscarriage at the third month, due to a fall downstairs. Following this the uterus came down and protruded from the vulva at least two inches, she thinks. A doctor applied a tight pad and binder and treated her for weeks. The uterus never came so low again, though she was often bothered with it. She wore a pessary for six months following the birth of the fourth child, fifteen years ago. This seemed to be of value in keeping the uterus in place. After the birth of her fifth child a doctor told her that her rectum protruded into the vagina badly. For twelve years she has not noticed the uterus abnormally low in the pelvis and has not had the dull, dragging pains that she formerly had in such a marked degree. Her bowels have been sluggish all her life, but much more so since her fifth child was born. During the past three years they have been very sluggish and she never has a movement without catharsis or the use of an enema. Temperature and urine normal.

COMMENTS AND OPERATION

DR. MURPHY: What is the matter with the woman?

THE INTERN: That history does not tell what is the matter with her.

DR. MURPHY: Important facts are left out. First, when she was examined by a doctor some three years ago he found her

uterus was low. Since then she has reached the menopause, and since the menopause she has had atrophy of the uterus, so that now it is not larger than my thumb, and it is up in fairly good position. She does not have procidentia uteri. The uterus is in an anteflexed position. When she goes to stool the rectum bulges out of the vagina. She has a laceration of the perineum, with prolapse of the rectum. There is no prolapse of the anterior wall of the vagina. She has no vesicocele and no procidentia.

In considering these cases the other day we divided the treatment into four kinds: first, cure by repair of the perineum, with reproduction of anteversion. Second, ventrofixation as a part of the treatment. Third, implantation into the anterior abdominal wall, and, fourth, extraventral splitting of the uterus so as to hold the vagina and rectum in place. The last three methods cannot be considered in this case, because it is only the prolapse of the rectum that is troubling her. She wants repair of the levator ani muscles and closure of the large vaginal opening below, so that when she strains she can have stool without the anterior wall of the rectum coming down between the labia. That tells the story. The history should tell this. The patient has nocturnal urination. What does this patient want to be relieved of? She wants to be relieved of the projecting forward of the posterior wall of the vagina and the anterior wall of the rectum. That tells the story. [The patient is in the lithotomy position.] Look at the rectum, coming right out without any straining at all. When she strains, the rectum protrudes more than it does now. There is no vesical prolapse at all.

We must expose the levator ani muscles higher up, in order to get them fixed. If you do an ordinary perineorrhaphy in a case like this, you would not get the desired result. If you do the old-fashioned operation of repairing skin and mucosa, the result will be much better. She has a very good perineum now, just as good as some women get sometimes from a perineorrhaphy. Lawson Tait is the man who really put perineorrhaphy on the proper basis. What we want to do here is to restore the muscular support.

We will make a U-shaped incision at the introitus vaginæ, encircling it and extending from caruncle to caruncle on either side to where the normal vaginal opening should be. I will then burrow upward under the vaginal mucosa until I reach the levator ani muscle. I want to keep under the mucosa and on the muscularis; I spread my scissors so as to separate the fascia without danger of producing a severe hemorrhage. The gauze-covered finger is also very useful.

Visiting Physician: How many stitches do you put in the levator ani muscle?

Dr. Murphy: About five, as a rule.

Visiting Physician: Do you use the figure-of-8 stitch?

Dr. Murphy: No, not at all. Now I come to the rectal sphincter and I pick that up. The sphincter ani has not been divided. We will now unite the torn levator ani muscles and fascia with catgut so as to give her a firm perineal body, which will also prevent the rectum from bulging through the vaginal wall. We have in all our stitches to support the perineum proper. We will then put in a loose subcutaneous and submucous stitch that goes across from one side to the other just beneath the vaginal mucosa. The vaginal mucosa will fall in over that stitch and bury it. This is essentially a Tait operation. It is a muscular approximation. You get these two levator ani muscles coming together. There is the real perineum. I will pass these stitches through the under portion of the anterior vaginal flap or the inner portion of the mucous flap of the posterior wall of the vagina for the purpose of obliterating that dead space in which occasionally a clot forms and which acts as a decided retardation to union. There is accurate apposition of the full length of the levator ani muscle clear down to the central raphé. The next thing is to put in a subcutaneous stitch. The perineum is now an inch and a half thick. If that holds,—and we have every reason to believe it will,—we will have a good and serviceable perineum, and there will be no further descensus of the anterior wall of the rectum. That is a submucous stitch, and finally, as we go a little farther down, it becomes a subcutaneous stitch.

This patient will receive lysol vaginal douches twice a day. When she wishes to urinate a soft catheter will be used and after each urination she will have the perineum washed off with lysol. The only thing which will interfere with perfect union is sepsis; therefore we must guard against that.

[The final result in this case was all that could be wished for. —Ed.]

UNUNITED FRACTURE, SHAFT OF RIGHT HUMERUS

HISTORY

MALE, railway brakeman, aged twenty-seven. Entered hospital because of an ununited fracture of shaft of right humerus. Denies syphilis in both self and family.

February 5, 1908, he was riding in the caboose of a freight train when a rear-end collision occurred. The car in which he was riding was completely demolished. He was rendered unconscious and remained so for fully three days. He received many injuries, all of which healed without incident except what he says was a compound fracture of the shaft of the right humerus. Two weeks after the accident his arm was taken from the wire splint in which it had been placed. The patient is not sure that the discharge had ceased, but knows that there was never much drainage. At this time the fragments were wired. Six weeks later the bone was still ununited. Wire was removed and he carried his arm in a cast. Three months after the accident he was operated on again. He thinks the callus was cut away and bone-ends were united with catgut. This was four years ago, and a false point of motion still remains at lower third of the right humerus.

Temperature, normal. Urine, normal. Hemoglobin, 80 per cent. Leukocyte count, 10,000.

COMMENTS AND OPERATION

DR. MURPHY: This man gave me to understand that there was a fragment of wire in there. He told me they took out a part of the wire, but did not take out the other part. Here is a typical pseudo-arthrosis with a good lining for the joint. Nature made a good artificial joint. We have been learning to make

them where we need them—in the knee, elbow, hip, etc. The fracture was when?

THE INTERN: February 5, 1908, four years ago.

DR. MURPHY: How many operations has he had?

THE INTERN: Three.

DR. MURPHY: It was a simple fracture to begin with; it does not look in the skiagram as though it were a compound fracture. What was the original treatment?

THE INTERN: It was put in a wire splint and left for nine days.

DR. MURPHY: What then?

THE INTERN: It healed completely in two weeks.

DR. MURPHY: We have pointed out repeatedly in our teaching that the reason the bone-plate and mechanical internal appliances are going into disuse is because they are put in open compound wounds. They must not be put in until such wounds have entirely healed and are aseptic beyond question. Every practitioner seems to be putting in a metal bone-plate. The steel plate of Lane must be put in with brains, and it must be put in under proper aseptic precautions, and it must be put in a field that is absolutely aseptic beyond question. That is what is happening with Lane's metal plate; that is what is going to happen with the bone-plate because to keep it aseptic is far more difficult than is the case with the Lane plate. I know of at least six cases in which the Lane plates had to be removed. Why? Because in all six of the cases the plates were put in under improper conditions. The failure is not to be charged to the Lane plate, but to the practitioners who undertake to do this work without getting all the facts and reading carefully between the lines, and who do not know how to do the work in an aseptic manner, after they have read the lines and between the lines. Even then we are apt to fail sometimes. Let us keep that in mind. Lane's plate was never intended to be inserted in a compound fracture until the wound had entirely healed and was altogether aseptic. Neither wires nor nails are to be put in in cases of compound fracture, because they have to be taken out, and the great majority

Fig. 124.—Photograph showing position of arm on attempted flexion of the elbow-joint. A typical false joint was formed at the seat of fracture in the lower third of the humerus.

have to be taken out before there is union. That has been our clinical experience, which has been enormous.

Regardless of what the etiologic factors were, non-union is likely to result if a plate or other foreign body is put in when the indications are not suitable for it. The nearer one is to the elbow-joint, the more unpromising the possibility of union is, because it is practically impossible to immobilize that elbow-joint at that particular point and prevent the brachialis anticus from contracting and prevent the extensor and flexor muscles of the forearm, that are attached to the condyles and condyloid ridge, from contracting and pulling that downward. When you straighten the forearm, see what happens! The brachialis anticus and the flexors and extensors of the forearm pull the lower fragment clear around at right angles. That is the difficulty we encounter in these cases. The implant in this case has been worked out of the lower fragment and pushed up into the upper fragment.

It was a bovine implant, and does not perform the function that an implant from the patient's own bone does. The capillary vessels of the human bone never pass into the Haversian vessels of the bovine or animal bone and carry with them the osteoblasts necessary for the regeneration of bone. We have learned that by experience. We did not know it when this method of splinting was first proposed, but at the same time we thought that it would give the mechanical support necessary, as ivory does, and that we would get union around the implant, although the implant acted as dead aseptic material.

In operating on this man now we are going to use an implant from his own tibia, which is the proper thing to do. We always take an implant from the same individual, and the best material to take is the compact bony tissue of the crest of the tibia. The crest of the tibia gives you the greatest latitude. Here we must put in a long implant that will go away down in the lower fragment, so that it will mechanically counteract the contraction of the muscles of the forearm from the moment it is put in, and it must be strong enough. I will make a magnesium metal nail which I may need in this case later. Magnesium is absorbable,

and as it absorbs it produces a considerable quantity of gas, so that it blows up and balloons the inner lining of the wound. You may need to aspirate and remove it if the accumulation of gas gets too large. You can hasten the absorption by passing through this wound any acid solution, but the rule is that it absorbs quickly without such aid. The tissue fluids absorb the magnesium very well.

Our plan of procedure in this case will be, first, to expose the fragments and place them in proper position. There are fixation and shortening of the anterior elbow-joint capsule. In making our incision, what have we to bear in mind? We have to remember the musculospiral nerve. I will make my incision in front, right at the margin of the biceps muscle. I am going near the anterior incision that was made here in the previous operation, so as to get near the same line of injury to the subcutaneous tissues. The upper end of the lower fragment is thus easily exposed. A beautiful capsule has developed around this false joint, showing how nature does the work. How nicely developed the capsule is! It is like the normal capsule of a joint, although this is a pseudo-arthrosis. There is fibrous union of the fragments. We will liberate the capsule from the fragments so that the bone-ends will be exposed freely. The anatomic relations are essentially destroyed, and what we have to do is to keep close on the periosteum all the time, so as to keep out of trouble. It is these new ligaments that have developed in this pseudo-arthrosis that are giving me the trouble now.

The cartilaginous surface of the new joint is fairly well developed. We must take all that out because we want to get accurate contacting of bone to bone in order to get bony union. The fragments come in accurate apposition without difficulty. I will remove the cartilaginous covering of the lower fragment and of the upper fragment so as to have a raw surface of bone for an end-to-end union.

We are now ready to get the bone implant, which we will take from the crest of the tibia in the usual manner. The fragment will go into the medullary canal of the upper fragment quite a distance—an inch and a half or two inches. We will enlarge the

Fig. 125.—Radiogram showing lateral view with lower fragment tipped forward above. Ends of both fragments are covered with an abundant callus, but with no attempt at union. Typical pseudo-arthrosis.

canal with our medulla reamer so as to receive the implant. The lower fragment will also be reamed out, but for a shorter distance than the upper.

In taking off this piece of tibia we never make the incision over the compact bone, but over the tibialis anticus, so that the line of scar will not be over the compact bony tissue; then we go in through the tibialis anticus onto the periosteum. We do not know that that periosteum has any value. In fact, from the pictures we have had, it looks as though it is a detriment at this age. In children it is an advantage.

I will remove a piece of bone about four inches long, and 5/8 by 3/8 inch thick and wide. A small drill hole above and below will mark the length of the implant. That guides my instrument in cutting the bone. The retractor guide which I devised will guide my chisel in cutting out the piece of bone and also keep the soft tissues away from the chisel, so that they will not be injured. This retractor guide has two short prongs, one on either end. These two prongs go into the drill-holes I have made and hold all the tissues back out of my field. I am going to saw the bone crosswise at the point where I made the drill-holes, because that is where I intend to fracture it. I take out a V-shaped piece of bone all along the edge of the retractor and then the bone will fracture in that line. If we hurry, without taking out a sufficient V, we may fracture the fragment crosswise. That deficiency in the tibia fills up readily. The implant is held with forceps, and when removed is carefully placed in a piece of sterile gauze so that it will not be touched by hands or instruments and remain aseptic. We have the fragment ready to be inserted.

The incision in the leg is closed with deep catgut sutures, the muscle being sutured over the trough left in the tibia after the removal of the implant. The skin is sutured with horsehair. The wound is dusted with bismuth subiodid, a desiccating powder, and sealed with gauze saturated with collodion.

Let the records show that we took out a piece of bone four inches long by five-eighths by three-eighths, triangular in shape. In making the incision we came down on the musculospiral nerve, displaced one-half inch inward by contrac-

tion of the cicatricial tissue. We were sure it was the musculospiral nerve. We kept it in view all the time during operation so as not to injure it. After taking off the capsule of the pseudo-arthrosis, which capsule was firm, compact, and hard, resembling the normal capsule of the joint with synovial membrane (cicatricial), the ends of the fragments were exposed, both covered with cartilage. The cartilage was entirely removed and the ends of the bones freshened. Then with the reamer a hole two inches long was made in the medullary canal of the upper fragment, and another an inch and a half long in the lower fragment. The ends of the fragment of the tibia were rounded off to fit into these holes in the fragments. One end of the implant was driven into the upper fragment, and with a little careful manipulation it was finally, with as little tension on the lower fragment as possible, adapted and slid into the lower fragment; then it was driven back into the lower fragment by a rectangular bone-crushing forceps which grasped the implant on the side. This implant establishes a firm union of the two fragments and will play its rôle in the regeneration of bone, which will proceed from the upper and lower fragments through the implant. The implant is finally absorbed.

A test made of the musculospiral nerve with the faradic current at the completion of the operation showed the nerve intact and not disturbed by the operation.

The deeper structures are united with catgut. The skin is closed with horsehair. The arm will be put in a plaster-of-Paris cast reaching up over the shoulder. The arm is flexed and fixed at right angles in front of the body. The cast extends well down on the wrist and firmly up against the shoulder, including the shoulder, that is, over the cap of the shoulder, clear up into the neck. The arm must be kept in that position for seven weeks, perfectly still, until the bone fragment is filled with capillary vessels from above and below, and until the new bone spans the gap across the line of separation. First, there is a revascularization of the Haversian canals in the implant. That is, they are filled with Haversian vessels from the upper and lower fragments, which carry osteoblasts and osteoclasts through the

Fig. 126.—Radiogram taken five months after transplantation of piece of tibia into humerus. Firm bony union with regeneration of bone proceeding from both upper and lower fragments. Patient has full use of arm, although complete extension is still impossible.

Haversian canals in the implant. Finally, as bone regeneration progresses there will be in the center living bone with a shell of dead bone outside, and you have a bony union long before the dead piece of bone is removed by absorption, but all dead bone is finally taken away. These Haversian loops anastomose, and the new lamellæ are incased in the old lamellæ. The osteoclasts take out a brick or cell, and the osteoblasts put in a new one. The only rôle which the implant plays is that of a scaffolding for the Haversian vessels with their osteoblasts and osteoclasts.

[The progress of this case was uneventful and finally terminated in complete bony union and an arm anatomically and functionally perfect. Bone regeneration progressed rapidly, as shown in the skiagram.—Ed.]

OSTEITIS FIBROSA CYSTICA OF RIGHT HUMERUS

HISTORY

SCHOOL-BOY, thirteen years old, entered Mercy Hospital because of lameness and tenderness of right upper arm. His family history is negative. He was always free from digestive disorders of all kinds; never had rickets, scarlet fever, rheumatism, tonsillitis, or sore throat. He always was a strong, active boy, with full use of both arms, and both arms and shoulders were of the same conformation.

Early in 1911, while throwing a ball very hard, he experienced a sudden pain in the right arm, four inches below the shoulder. On attempting to raise the arm he found that he could not do so; it hung limply by his side. A doctor told him that it was broken, and placed the arm in a splint for six weeks. Union was apparently perfect, and he gradually regained full use of the arm.

In March, 1911, he fell on a stairway, striking on his right arm and breaking it. He carried the arm in a splint for six weeks, and again it became perfectly useful, but for four or five days only. He fell to the floor, striking on his left shoulder, but the right arm broke (third fracture). There was little pain connected with the injury. The arm became useful again, though it remained a little lame.

In July, 1911, he attempted to sit down on the floor and felt his arm and hand were limp. He had no pain. The arm was not fractured, but on examination he noticed for the first time that the arm was larger at point of fracture and uneven. All the fractures had occurred at the same point. Arm again became useful. Four weeks ago arm was lame and he could not raise it from body, and a tenderness has been present at site of fracture since that time. Wassermann negative. Leukocytes,

8200. Hemoglobin, 80 per cent. Urine alkaline, but otherwise normal.

COMMENTS AND OPERATION BY DR. JOHN F. GOLDEN

Incision was made directly over point of right shoulder on its outer and slightly on its anterior aspect. This divided the deltoid longitudinally and passed down to the bone, the shaft of the humerus, for fully six inches. At the upper angle of the incision the capsule of the joint was exposed, and at the lower angle could be seen the musculospiral nerve. This nerve was tested with the excitor both before and at the completion of the operation and found to be uninjured.

At a point 5¾ inches below the head of humerus the shaft showed absolutely no signs of involvement. Here the bone was freed of all its attachments, retracting the musculospiral nerve. A chain-saw was placed about the humerus at this point and the bone was divided.

The capsule of the joint was now divided circularly at its attachment to the neck of the humerus. The muscular attachments about the head were freed, keeping very close to the bone and even removing a small amount of it at the greater and lesser tuberosities. The outer head of the biceps was lifted from its place in the bicipital groove and displaced inward. It was not cut.

Now the upper end of the humerus was plainly visible. This portion was fully 5¾ inches in length and triangular in form, with broad end at upper end of bone. The largest diameter was 2½ inches. The mass was globular, expansible, and cystic, with eggshell-like portions of bone in places, and brittle. In other places it was more fibrous and less fragile. The periosteum had previously been removed when the muscles were taken off. Periosteum was carefully examined and found to be free from involvement in the process. Beginning at the inner side, the periosteum was elevated and the mass rolled outward and upward, detaching the periosteum from it until it was completely freed. Great care was used lest the musculospiral nerve be endangered in this process. The head of the humerus was not involved

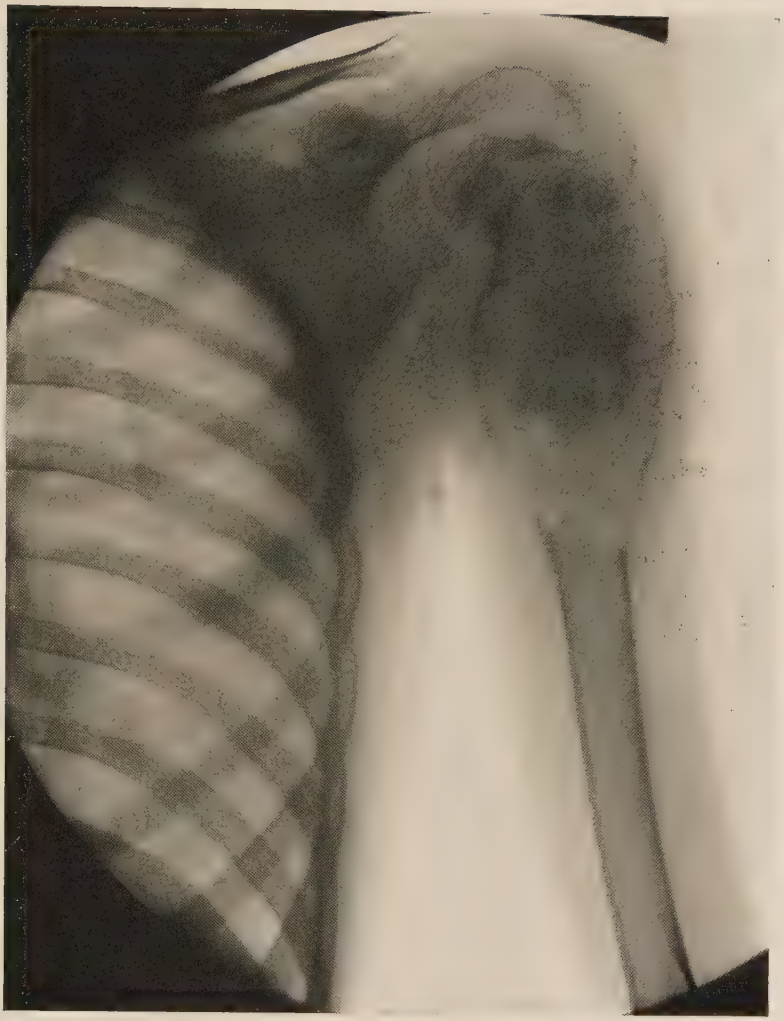

Fig. 127.—Skiagram showing case of osteitis fibrosa cystica of upper extremity of humerus before operation.

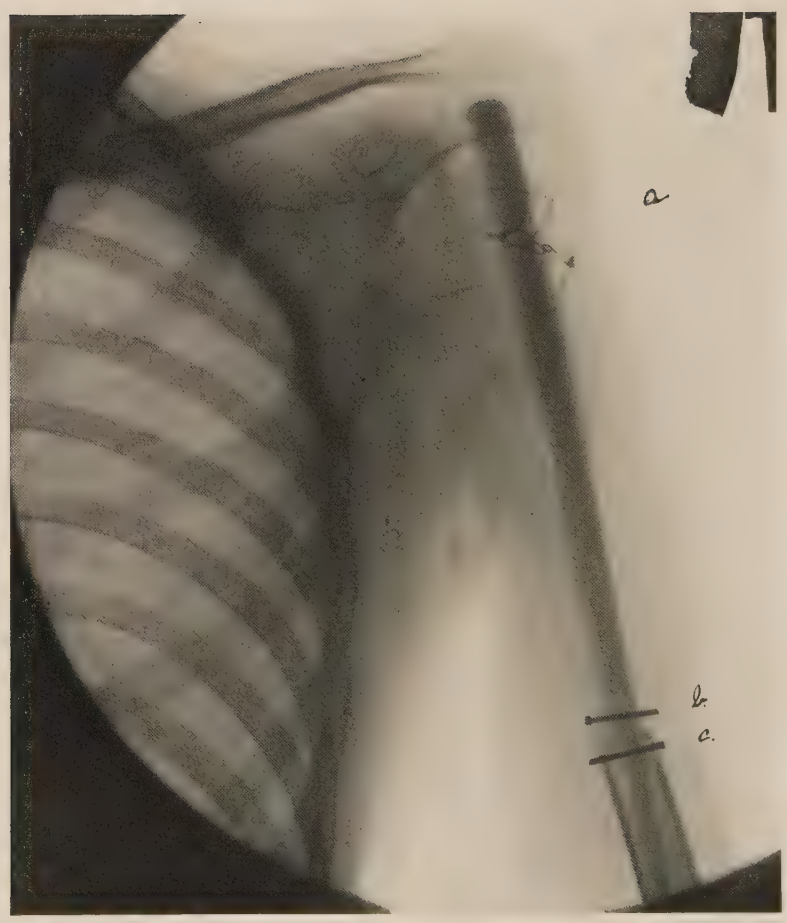

Fig. 128.—Skiagram showing tumor removed and tibial transplant in place. The lower end is inserted into the medulla of the humerus shaft and held by two nails driven through both bones. The upper end is in the glenoid cavity. The muscles are sutured around the transplant with phosphor bronze wire.

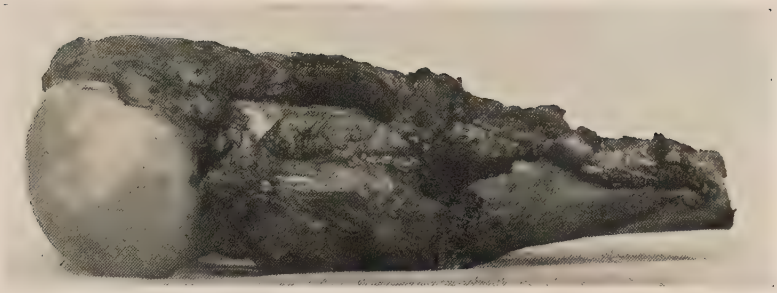

Fig. 129.—Photograph of removed diseased bone, showing cystic condition of bone above and normal shaft below, where musculospiral nerve crossed (view 1).

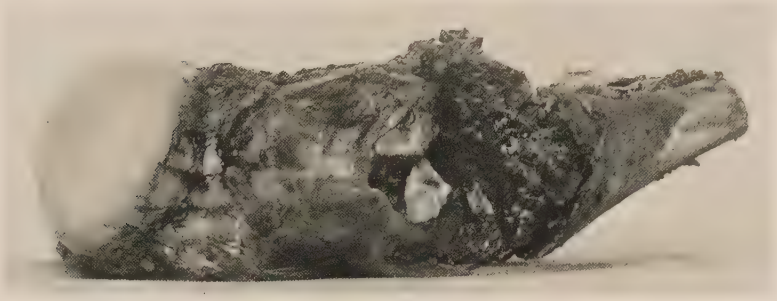

Fig. 130.—Photograph of removed diseased bone, showing cystic condition of bone with wall purposely broken in to demonstrate shell (view 2).

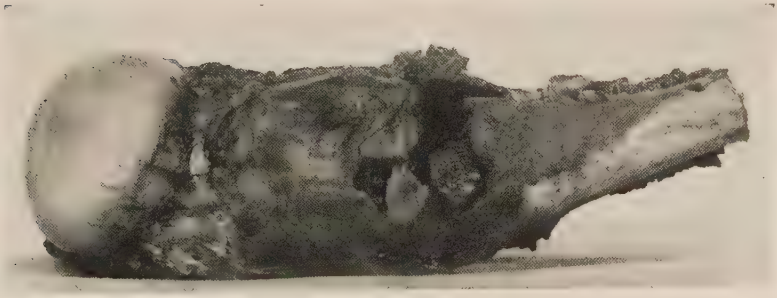

Fig. 131.—Photograph of removed diseased bone, showing same as view 2.

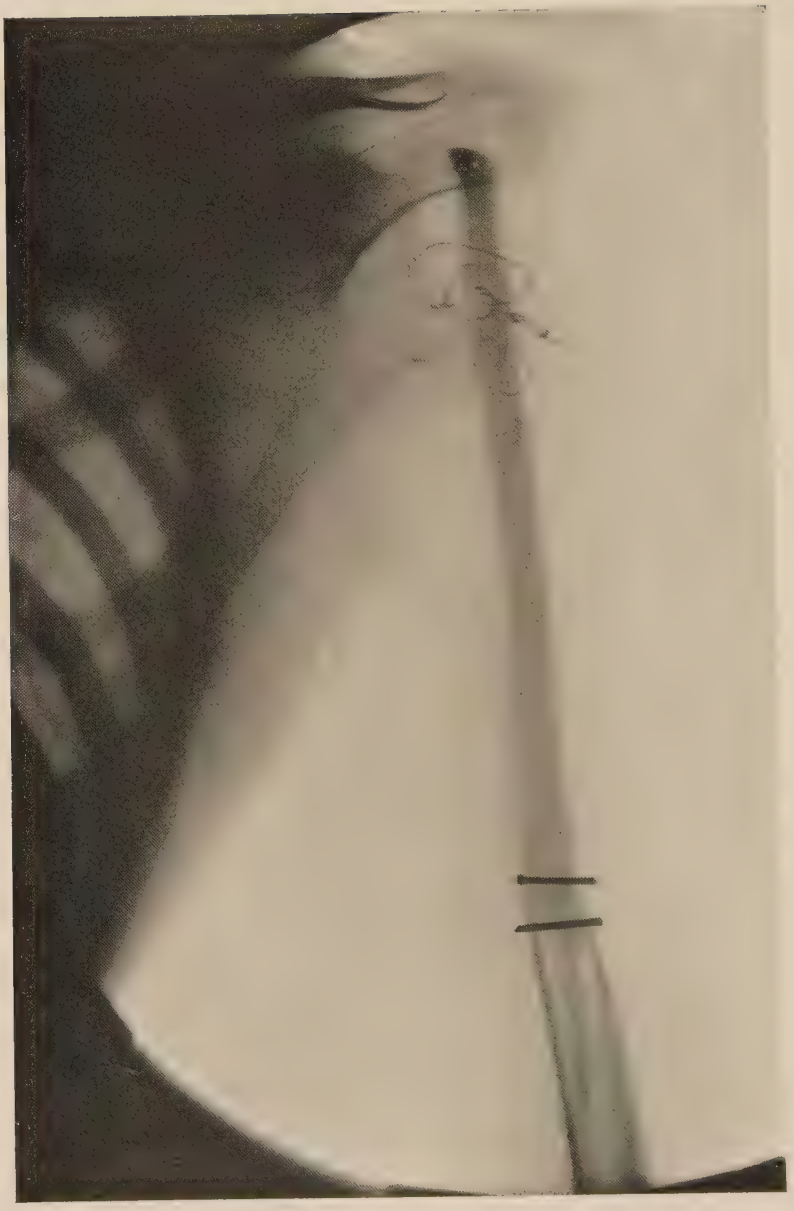

Fig. 132.—Skiagram of transplant five months after operation, showing regeneration of bone progressing upward toward joint from shaft of humerus. The new bone is easily visible along the edges of the transplant.

in the process, and the articular surface was apparently perfectly normal.

It was a question whether or not to remove this head. It finally was decided to remove it.

A transplant seven inches in length was now removed from the right tibia and ends rounded into shape. A hole was drilled in each end of this strip, the lower hole being about 1½ inches from the end. A hole was also drilled in the medulla of the shaft of the humerus, and the transplant inserted into this hole a distance of 1½ inches. A nail was placed transversely through transplant and humerus, thus preventing the transplant becoming further embedded in the medullary canal. Using a catgut suture as a purse-string, the capsule of joint was puckered in about the upper end of the transplant, which rested in the glenoid cavity. Now two wire sutures were passed through a hole in the upper end of the transplant and also through the joint capsule. Muscular attachments were not included in this. The wires were tied about the capsule only as an additional means of keeping the transplant in position in the glenoid fossa. The old periosteum of the humerus was sutured around the transplant. This carried the muscular attachments, none of which had been separated from it. A row of sutures closed the incisions in the fascia and aponeurosis; another of horsehair closed the skin incision. A plaster cast was applied, covering the arm from the side of the neck to the wrist, with arm at right angles.

[*Note.*—Six months after operation: Patient made perfect recovery and has now full function of arm and a movable shoulder-joint. The improvement is very satisfactory. The regeneration of the upper end of the humerus is progressing well, proceeding upward from the shaft of the bone.—Ed.]

ANKYLOSIS OF LEFT ELBOW

HISTORY

MALE, aged thirty-one, single, salesman. Came to hospital because of ankylosis of left elbow. Childhood disease with good recoveries. Typhoid fifteen years ago, also with good recovery. No tonsillitis, alveolar abscess, discharging ears, or enlarged glands in neck. Patient has been in perfect health up to the time of present trouble.

About May 1, 1911, patient was exposed to Neisserian infection; four days later the discharge from urethra appeared. He first noticed joint trouble in the right wrist. He states he did not injure his wrist. Pain, redness, and a little swelling and inability to move the wrist without causing intense pain were the chief symptoms. Wrist trouble cleared up in six weeks from onset. Three days after the onset of pain in the wrist the left elbow became involved. For eight weeks he kept the elbow at rest because of the pain, at the end of which time the elbow-joint was stiff. He had massage, passive motion, electric baths, and finally took gas to have the adhesions broken down. Arm was dressed in plaster cast and ankylosis recurred. Did not have a chill at any time.

COMMENTS AND OPERATION

DR. MURPHY: That is a fairly classic history. The patient denies having had a chill. The history does not state as to the stormy symptoms that preceded the metastatic deposit of the Neisserian microörganism in his elbow-joint. He had a mild urethritis, a specific urethritis, a purulent discharge manifesting itself four days after exposure to the infection. He went on for thirty days before he began to have pain in his wrist. That pain was not very severe or stormy; it was rather a mild mani-

festation of the metastatic joint infection, which is the exception. The elbow trouble began with a definite onset because he remembers accurately the number of days before he had pain in the elbow. His wrist recovered, which is common when you have arthritis as a metastatic manifestation of a Neisserian infection. His wrist got perfectly well and remained perfectly well. The inflammatory condition concentrated itself in the elbow-joint. This joint was immobilized during the acute stage of the lesion, and the result was a complete ankylosis. This was broken up under anesthesia and the arm put into a cast, again resulting in a bony ankylosis.

Let us follow the history and see where it deviates from the usual course. The rule is that in a metastatic arthritis from a urethritis metastases take place in the joint between the eighteenth and twenty-second day in 96 per cent. of the cases. He says that it was thirty days before he noticed the trouble. That is eight days after the usual time. That is not an accident. Frequently patients will say, "I got that because I was exposed in my automobile," "I got that because I got my feet wet," or "I got that because I was in a draft and had a chill," when the fact is that the chill is the manifestation of the metastasis. All types of arthritis are metastatic manifestations of infection in other parts of the body. I am speaking of acute arthritis. There is not a particle of doubt in my mind about that. It is an opinion based on a colossal experience, with careful analyses of the histories of the cases. The reason we have failed to find the primary lesion in the majority of cases with metastatic lesions is that the metastasis does not take place until after the primary lesion is forgotten. The patient was up and moving about when he got the "rheumatism." That is such a common expression, "Then I got the rheumatism." I recall the time very well when we had general sepsis in our hospitals, and with the metastatic joint inflammation came on pyemic symptoms. Exactly the same relations exist between arthritis and inflammation and infection of the mucous membranes and mucous cavities, like the gall-bladder, nose, throat, and tonsils, except that the metastatic infection is of the mild type and does not produce

Fig. 133.—Complete bony ankylosis of radius to ulna and radius and ulna to humerus, with obliteration of joint anatomy.

pus in the joints. This is so significant, and so positive is my conviction on that score, that I mention the fact at every clinic, because on that basis every one of these cases is primarily a surgical case.

A joint lesion is a metastatic infection, and the final result depends on the virulence of the infecting microörganism and the management of that joint lesion. If these cases are handled properly, you can avoid all the frightful deformities so often seen by placing the limb in good position and maintaining that position during the inflammatory process. If you let the limb get out of position in the first few days, you will never get it straight. If you put the limb in good position with extension immediately, you will not only relieve the pain but prevent deformity and in all probability avoid an ankylosis.

Another matter that led to our undoing in this class of cases was aspirating these joints. We did not find any infectious material in the joint, and we therefore concluded that it could not be metastases. You may remember the extended and heated discussion we had some years ago as to whether certain joint infections were gonorrheal or not. One observer, say, examined one hundred cases and found gonococci in seven. That was looked on as a coincidence. Another man would examine the cases in another stage, and in 90 per cent. he found the Neisserian microörganism. It all depended on the time or stage of the affection that the examination was made as to whether or not any cocci were found. But it was always a secondary lesion, a metastatic lesion, with a definite, unvarying clinical course.

There is not a particle of motion in this elbow-joint. I will make the skin incision on the back of the arm, slightly to the inner side of the midline of the olecranon—an incision 5½ inches long, extending down to the bone, so as to give me free access to the joint. I am figuring for my interposing flap. You have to be governed by the amount of fat deposited. I am leaving all the fat on the aponeurosis of the muscle. I will take off the aponeurosis of the triceps muscle and interpose it later between the olecranon and the humerus when I separate these bones.

I will also use a portion of the capsule of this joint for that purpose. The aponeurosis is rather a dense fibrous mass. It is the type of fascia one occasionally finds between the ends of fractures and results in a failure of union. That knowledge led me to do this arthroplasty operation originally.

I will take the aponeurosis with the fat. The preparation of this interposing flap is the most important part of the procedure. I have made this external incision long enough so that it exposes the triceps. The fibers of the triceps and of the supinator longus, as well as the attachment of the long flexors of the fingers, are plainly visible. The olecranon, the tendon of the triceps, the external condyle of the humerus, are landmarks which are now in view.

The ulnar nerve is the one nerve that is likely to be injured in this procedure, therefore we will expose it to view by making another incision on the inner side of the elbow, parallel to the incision on the outer side. I do not wish to handle or compress the nerve during any stage of the operation, because in that way I could seriously interfere with immediate transmission of impulses by traumatizing its axons. With the nerve standing out very clearly and distinctly, we can keep it out of harm's way. I can now separate the radius and ulna and humerus from each other without in any way hazarding the ulnar nerve. It will not be interfered with at all.

Now we are going to separate the bony union. There is an ankylosis in this case between the ulna and the radius, as well as between the radius and the humerus. There is not a particle of motion. The line of union between the humerus and the ulna is easily located. The head of the radius is difficult to make out. Having definitely located the neck of the radius, I will remove the head of the bone, taking with it the periosteum, as if we leave the periosteum in place here it will be a great detriment to the restoration of motion subsequently. I chisel the radius free from the ulna, and then remove the head of the bone, about one inch, with bone-cutting forceps. That separates these two bones well, and leaves things at about their normal conformation.

Fig. 134.—Skiagram taken four weeks after operation. Head of radius was removed. The cleft between the olecranon and the humerus shows that the interposing fat and fascia flap fulfilled its function. The motion in the joint was returning rapidly.

Now we must get the ulna freed from the humerus, which is not at all so easy a task. Now you see the advantage in having that ulnar nerve out of harm's way. You can see how well I have protected that; I can cut in here between the coronoid process of the ulna and the humerus in order to make my bone division without hazarding this nerve in the least. We will carefully chisel free the olecranon from the humerus, so that we can look right through the joint from one side to the other.

Now we have the bone free without having fractured it. We next slowly stretch the soft parts. Having removed the head of the radius, we have reëstablished pronation and supination. Pronation and supination are very important matters in these cases. The next step is to remove enough of the coronoid process and of the humerus to permit the interposition of the fat and fascia flap.

With chisel and forceps we have changed the conformation of the lower end of the humerus to about normal. I will be ready for my interposing flap after I have taken off the necessary amount of the coronoid process in front. That is one of the greatest annoyances in this work. That coronoid process must be removed, separating the tissues in front gently and leaving the periosteum on the bone. I shall remove the periosteum of the coronoid process, otherwise it will reproduce bone there, and that is a very great hindrance in the subsequent course of the case. You can see how easily I have separated the coronoid process from all its structures in front all the way across. You have a full view of the conformation of the new joint.

I did not prepare this flap until I had all my work done, to see just what style of flap I would need. I take this flap from the aponeurosis of the triceps, base directed upward, and the flap long enough so that it will swing into position between the olecranon, articulating surface of ulna, and internal condyle of humerus. I will pull this flap between these bones and suture it in place with catgut. A similar flap is brought in from the outer side, covering the external condyle. There is perfect interposition between these bones. We throw the ulnar nerve back into position; we are all the time keeping the ulnar nerve out of harm's way and protecting it. See how nicely that goes

back in place, not contacting with the bone, but with the internal fat and fascia flap which I placed under the nerve for the special purpose of preventing a recurrence of the ankylosis, and for the second purpose of taking care of that ulnar nerve.

When you look at this elbow now it does not seem as though we had done much. It looks as though we spent a lot of time doing very little work, and when you get the stitches placed there is little to show for the work.

One of the greatest dangers in all this work is the danger of infection, as we handle so many instruments and do so much manipulating of the joint structures. We have been fortunate—I say fortunate, because no one has a supreme confidence in his asepsis except those who have had little experience. Up to date we have been fortunate in these cases in not having much infection. We had one man with sepsis of the hip; it was the second hip arthroplasty done for a double ankylosis of the hip. He got the infection on the tenth or eleventh day. He had been a morphin victim. He was not getting enough morphin, so one day he tore his dressings loose in the night and scratched the wound with his cane. He got an infection and died three weeks later. This is the only death we have had. We have had mild degrees of sepsis in other cases.

There is some oozing of blood, but I think it is only a venous oozing, and with the ordinary dressing pressure all that will subside. We rarely ever have to put on a ligature to stop hemorrhage in joint work. There is always some oozing from the surface of the bone that was divided.

This gives us now full and complete rotation, pronation, and supination. We divided the attachment of the ulna, its olecranon and coronoid process, from the humerus, which was the bony ankylosis. This was done with the curved and straight chisel and was not difficult. When it was free, a considerable portion of the tip of the olecranon was removed and a portion of the coronoid process well down toward its base. Then the intercondyloid groove and the trochlea were enlarged, and a portion of the end of the humerus removed, including both the condyles and the intercondyloid structures. The internal flap was interposed between the internal condyle and the olecranon and the

base of the triceps tendon. The external flap covers the external condyle, passed between the radius and the humerus on the end and between the radius and the ulna on the side.

The technic of this operation varies from our original technic in this: In the original technic we divided the olecranon from the shaft of the ulna. Now we do not do that. We leave the olecranon intact all the time. It has this advantage: that early passive motion can be instituted, and that is a very important factor. We have not had, since we have followed this plan, any sloughing of the skin-flap over the olecranon and the passive motion will not hazard that interposing flap at all, will not put the line of union materially on the stretch, so we can start passive motion very early and get a more early movement.

The next and last step is to suture the skin incision with horsehair. I am forcing out the blood that is just beneath the skin so as to get it empty before we put on our compress. We want as little blood in there as we can possibly have, and now is the time to force out that blood. That acts not only as a sealing device, but it mobilizes or splints the skin, helps to keep it steady, and supports the line of union. We dust the wound well with bismuth subiodid powder and seal it with collodion gauze. This is the carbolated gauze you have heard me speak of before. That carbolated gauze has the advantage, as you know, of caring for the flap in an antiseptic way. It is placed over the collodion gauze. That dressing is quite loosely applied. We have no constricting dressings placed on here. We dress that arm at just a little less than at a right angle, so as not to compress the interposing flaps. A plaster-of-Paris cast is then applied, and when it has set we split the cast on the lower side with a Gigli saw, to give some play, if there is any swelling of the arm subsequently. We want that arm dressed in the elevated position. All that you see is done with a definite purpose from the very beginning to the end of the operation, to get a proper interposition. The horsehair stitches will be removed in ten days or two weeks, and then passive motion will be begun. In three or four weeks the patient will be encouraged to bend the arm himself, so as to restore the normal function of the joint as soon as possible.

ANKYLOSIS OF RIGHT HIP-JOINT

HISTORY

SALESMAN, age twenty-one. Entered hospital because of ankylosis of right hip and limitation of movement in right knee-joint. Family and personal history negative up to time of present trouble. March 2, 1911, patient exposed himself to a venereal infection. March 7th, five days later, he noticed a creamy yellowish discharge from the urethra. After treatment for two weeks the discharge ceased. April 11, 1911, just nineteen days after the disappearance of the urethral discharge, he experienced slight pains in inner aspect of right thigh, six inches above the knee, not severe enough, however, to prevent his keeping social engagements on each of the three following nights, the last of which was for a dance. He distinctly recalls having become quite warm while dancing, and perspired enough to dampen his clothing. After the dance, at one in the morning, he drove two miles in an open automobile. There was snow on the ground, and it was quite cold; he became chilled. On reaching home, before he could get half undressed, an intense pain seized him in the right thigh, just where previous pains had been felt. He said it came without warning, and he was forced to lean against the bookcase for support. It persisted without abatement, and he hobbled over to the bed and had great difficulty in moving his limbs enough to get in under the bed-covers.

He is sure he had no chill and did not vomit, and thinks there was but little fever. The hip was very tender that day, and this persisted for weeks. It was not swollen, red, or painful, nor has it been since that time. As he lay in bed he never turned on that side. He was taken to the hospital the day following the dance, and a Buck's extension with a five-pound weight was applied to the leg. He protested vigorously against this, and says

he got the most ease by having the leg and hip partly flexed, with the thigh adducted, slightly throwing it over the other limb. He lay turned away from the diseased side with his weight mostly on the opposite side.

Two days later the extension was removed and a plaster cast was applied to the leg, extending from the axilla to the ankle. It was very tight and was placed entirely about the body and the right leg. It was not cut through to relieve tension, and caused him much pain. He wore this cast for fully five weeks, and large pressure sores formed on the heel and the back in the lumbar region, the latter extending for at least 6 inches directly over the spinous processes of the vertebræ. He lost 50 pounds in weight.

Seven weeks after onset of pain in thigh the tenderness in hip-joint still persisted. A doctor said the cavity of the hip-joint contained pus and advised an operation. The hip was laid open by an incision 5 inches long, and a large rubber drainage-tube was inserted, but the pus failed to appear. The tube remained *in situ* and the wound was open for two weeks.

Following this drainage operation the joint became completely ankylosed. In August, 1911, the physician attempted forcibly to flex the thigh under anesthesia. The flexion occurred all right, but the point of motion occurred below the joint, in the shaft of the femur, and today that limb shows over one inch of shortening.

Right knee cannot be flexed to more than an angle of 30 degrees. Right hip-joint is completely ankylosed. Upon examination of the heart a systolic blow was heard over the third left interspace. Apex-beat within nipple-line. Pulse irregular, rate averaging 66. Temperature normal; urine normal.

COMMENT AND OPERATION

Dr. Murphy: The discharge ceased nineteen days from the day he exposed himself.

The Intern: Two weeks, I think.

Dr. Murphy: No; the discharge ceased in nineteen days. The last Thursday in March, 1911, this patient exposed himself to venereal infection, and the Tuesday following, five days later,

he noticed a creamy yellow discharge from the urethra. After treatment by a doctor the discharge ceased. He was not treated before he noticed a discharge. He was not treated until he noticed it, so that the discharge ceased at the end of nineteen days. I will show you in a moment that every day counts and must be counted.

THE INTERN: Tuesday, April 11th, nineteen days after the appearance of the discharge, patient experienced a slight pain on the inner aspect of the right thigh.

DR. MURPHY: He was sure he had no chill after the pain began, but he was chilled and felt cold in going home from a dance.

THE INTERN: He did not have a chill, but he felt chilly and cold.

DR. MURPHY: What is the story now?

THE INTERN: A drain was put in.

DR. MURPHY: Did you insert the drain?

THE INTERN: I was told about a drain being inserted.

DR. MURPHY: The drain was kept in how long?

THE INTERN: It was kept in two weeks.

DR. MURPHY: And following the removal of the drain, what was his condition? He must tell the story. The examination is what we made afterward. What was the sequence of the disease? What was the sequence of the drainage? What was the sequence of the pressure? And why did he come here to be examined? That is what we want to know. He is here to be examined. He is unable to get motion in his hip-joint. The record does not show that that hip-joint is ankylosed.

THE INTERN: The examination does.

DR. MURPHY: That is not it. That is our opinion of it. That is not the history. The history is that from the time the drain was removed the joint was ankylosed and has been stiff. There was some effort made, as I understand, to get motion in the joint, according to the first record.

The history is a record of the patient's statements. I want you to have the record right, and the way to make a diagnosis is on the record, and on the record as given by the patient, particularly the order of facts. Now, we find out how nicely the order

of facts fits right into the pathologic condition, and why this man is here.

THE INTERN: In August, 1911, the thigh was flexed under anesthesia.

DR. MURPHY: The point of motion occurred below the hip-joint; he felt something snap when it was done, and the doctor believed he had a fracture. The skiagram does not show a deformity as the sequence of a fracture, but it shows there is some deformity resulting from the break. What date was that?

THE INTERN: August, 1911.

DR. MURPHY: That was a fracture of the neck. We can fracture it in the neck, throwing it out now, notwithstanding so long a time has elapsed since the injury. The story is what? It is a classic story. The dates are exactly right. He had a primary Neisserian exposure, and with the Neisserian exposure infection commenced immediately. Nineteen days after his exposure he had treatment in the regular way, but he got an additional exposure. He charges it to the ride home in an automobile and to exercise. Possibly that was it. I don't believe it, because he had pain in the hip preceding that ride, and he informed us that he was out in the rain that day, and the next day the joint was swollen. There was a second exposure, causing the metastasis to localize itself at one particular point or in multiple points in his joints. Here the metastasis took place in one joint. There is a peculiarity about the exposure when it attacks only one joint. No matter whether it is a Neisserian infection, tonsillitis, bronchitis, pharyngitis, or whatever the primary lesion may be, when the metastasis only attacks one joint, the attack is much more severe and much more likely to lead finally to bony ankylosis. One of the things that should be suspected immediately when only one joint is attacked is that it is going to result in an ankylosis.

Now, let us take up the management of the case.

Shortly after the manifestation of the joint trouble a Buck's extension was put on, but that did not give him relief, and the reason why it did not give relief was because the weight was not heavy enough. Five pounds is too little. You might as well

put on a postage-stamp. Buck's extension is put on for a purpose. It is put on with cortical cells, and not merely with weight and pulley and plaster. It is put on for the purpose of separating these two inflamed surfaces, and the weight must be sufficient to produce that separation. The weight must be enough to afford the relief that is necessary.

He was relieved after enough Buck's extension was applied. As far as Buck's extension is concerned, the treatment was right in principle, but wrong in execution. If they had put on 25 or 30 pounds, the man would have gone to sleep more readily than he would have done with a dose of morphin. He has an infection of the joint of Neisserian origin. Neisserian infection in 95 per cent. of all the cases takes place in the joint between the eighteenth and twenty-second day after the primary invasion of the urethra or mucous surface, and it always takes place at that time if the patient has a secondary extension of the inflammation in his urethra, as from sounding, or additional exposure or trauma. All these cases of so-called "rheumatism" are instances of metastatic infections. There is no such disease as idiopathic "rheumatism," any more than there is idiopathic peritonitis. They are all metastatic conditions. Here it is easy to trace the primary lesion, because his discharge from the urethra continues. Metastasis never takes place in streptococcic infection except after the primary infection. It takes place in from twenty to thirty days, depending on the virulence of the infection rather than on the resistance of the patient, if it takes place at all. That is why for so many years we failed to connect the primary infection with the metastatic infection. It comes after a long span of time has elapsed. In the urethra we can establish the connection because of the discharge continuing, but there must be a definite pathologic reason for the metastasis taking place between the eighteenth and twenty-second day. There is a definite law governing this just as there is a reason for the sun appearing every day.

At the last clinic we had a beautiful illustration of an osteomyelitic lesion resulting from furunculosis of the arm, showing the time the furunculosis was present. It was fourteen days before

the patient had her chill and a metastatic inflammation in her tibia, which caused the final necrosis.

In the management of this case, what happened? The joint was immobilized under great pressure. Wherever you have two serous surfaces inflamed, you must relieve the pressure. The synovial membrane is destroyed by two particular factors in infection of the joints. First, by the virulence of the microörganism that produces the synovitis; second, by the intra-articular pressure, by the involuntary contraction of the muscles forcing these two inflamed surfaces together. When we have an infection of a synovial membrane, if we would maintain separation of the inflamed surfaces; if we would avoid synechiæ, we must keep these surfaces apart. Here the surfaces were pressed together and the patient had pain all the time. He had not only pain from pressure in the joint, but he had pain from pressure of the mechanical support placed around his body.

We come to another proposition, namely, when you have infection of a serous surface, how are you going to maintain the integrity of that surface? *By reducing the virulence of the infection at the earliest possible moment, by relieving the pressure of the products of infection, and by keeping that surface away from the external air.* That is a law. If you expose any serous surface to the air for any considerable length of time, the endothelial cells lining that serous surface are *always* destroyed and synechiæ result. Once you decide on opening and draining the joint and maintaining drainage for any considerable time, the best result you can get, is what? An ankylosis. The worst result you can get, if you let it alone, is ankylosis.

Remember, these cases are impersonal, and what I say does not apply any more to this case than it does to others. My remarks are all impersonal; they do not apply to this particular case or to that particular case, but to the group of cases. One gentleman who attended my clinic went out and said that I criticized a certain man's work. Never! I criticized the principle involved. That is all I talk about. A man does his best with the knowledge of his time and the period in which he was educated.

That is all he is expected to do, and any criticism I may make is made on that basis and in that spirit.

What happened subsequently? After this joint had been immobilized it was drained, the discharge came out, and finally the tube was removed, and here comes the regular sequence, complete bony ankylosis, one of the greatest misfortunes that can occur to a joint. This man has complete union of the head of his femur with the acetabulum. This is the sequence of, first, infection and, second, drainage.

We will now proceed with the operation. We will do an arthroplasty in the usual manner. A U-shaped incision surrounds the great trochanter, the base of the flap being upward. I take out with this flap, the fascia lata, from which I will make a lining for this joint. This U-shaped flap is about three inches wide and five inches long. Formerly I made this flap longer, but I found that the flap occasionally died as a result of that, and so I have ceased to make it so long, but I make the deep flap long. The fascia lata and fat are dissected from the under surface of the flap, remaining attached at the upper end. This tissue flap will be turned in to cover the head of the femur and be interposed between it and the acetabulum, so as to prevent a recurrence of the ankylosis. We have taken off a double flap. It is a skin-flap, and an aponeurotic flap. The gluteus maximus is pushed out of the way, and here is the gluteus medius and there is the trochanter. We are going to saw off the trochanter with a chain saw and turn it with its muscular attachment upward out of the field of operation. I shall, if possible, try to get under the pyriformis muscle and the obturator muscular attachments. I leave all the muscles attached to the trochanter. That exposes the joint nicely. I will divide the capsule, open it up, and get ready for the excision. The capsule has become adherent around the neck of the femur. We will free it from its attachment. Now I am cutting the pyriformis muscle. The first time I cut this muscle I thought I had cut the sciatic nerve, as it is just like it, but not so far away. I am just leaving some of this tissue so that I can resuture the muscle.

Let the record show that I am freeing the capsule from the

neck and turning it out; I may utilize this capsule subsequently. This is slow work, separating the capsule from its attachments to the side of the neck of the femur. That exposes the line of union between the femur and the acetabulum. This is a case of bony ankylosis of short duration. It is of Neisserian origin. It should not be of the dense osseous type by this time. It should fracture easier. It should follow somewhat the normal conformation of the joint with a little effort on my part. Softening has taken place on the anterior side of the head of the femur more than on the superior. With a curved Buck chisel I will chisel out the head of the femur so as to maintain its normal rotundity. I have a fairly good head, about the right size, and about the right conformation. It came out just about right. It is a little elongated; that we must shorten because we will have to put in our soft tissue flap. We have very few special instruments in doing this work. You need no special armamentarium to do this work. We must deepen the cavity from which this bone came out. For that purpose we use an instrument called a reamer. It is attached to a brace and the acetabulum is reamed out to the desired depth. An end mill, which fits into the reamer, restores the normal conformation of the head of the femur. That gives us a normal hip-joint—both anatomically and functionally. The next step is to place the interposing flap between the head of the femur and the acetabulum, to prevent the recurrence of the ankylosis. This interposing flap is taken off from the gluteus maximus with the fascia lata, which I prepared in the beginning, with a long tail so as to have it sufficiently long to go all around the acetabulum. It is larger than my skin-flap.

Visiting Physician: What about the danger of necrosis of the flap?

Dr. Murphy: I do not know whether that flap dies or what happens to it. I do know that no union follows. I do not think it necrotizes. It is a connective-tissue flap, as you see, taken from the fascia lata, which has feeble vascularity. It is sutured in place with a phosphor-bronze wire suture. I am suturing this to the capsule on the anterior side, to the capsule

Fig. 135.—Skiagram taken before operation, showing complete bony ankylosis between head of femur and acetabulum with complete obliteration of all normal anatomy.

Fig. 136.—Skiagram taken immediately after operation, showing phosphor-bronze wire sutures in edges of interposed flap and wire nail transfixing trochanter.

on the posterior side, and to the capsule below. The bone will rest on that fat, muscle, and fascia, but principally the fat and fascia. You see how that is sutured to the margin of the acetabulum. These are just tacking stitches to keep all the raw bony surfaces covered. We learned in our pathology that if we interpose fascia or muscle between the two ends of the fragments of a fractured bone, you have failure of union. We have taken advantage of that pathologic knowledge by putting in these flaps, and it was on this basis that, ten years ago, we did our first arthroplasty. It was on this basis that we planned it, and this technic is identical with the original technic.

It was a hip-joint case on which I operated for the first time. We had occasion to demonstrate that case at Seattle before last summer. The patient was from Everett, Washington. I saw him nearly nine years after I did the first operation. He has a splendid anatomic result, with perfect motion in the hip. He rides horseback, and does anything that any young man of his age can do.

This flap is anchored all the way around to the capsule of the joint, so that it cannot become misplaced. You can put it on that way or suture it around the neck of the femur, which we have done, and turn it back. I like this plan—it is more precise and does not displace any of the restored fragments. We are ready for the restoration of the bone. We force all the blood out of this field. The head of the femur is easily placed back into the acetabulum by flexing the leg on the thigh and adducting it slightly. It stays perfectly in place. We suture the anterior portion of the joint capsule to what is known as the Y ligament, that caused so much discussion in the last decad of surgery. That immobilizes the bone. Every step of this operation we carried out on the cadaver before we attempted to do it on the human being. All our operations have been successful. The ankylosis has never recurred, and the normal anatomy and function of the joint have been restored. These patients never return for a second operation.

We have had many combinations of this operation that we do not count as true arthroplasties.

Now we are ready to do the next step, which is the fixation of the superior portion of the capsule to the acetabular margin with phosphor-bronze wire. That closes up the joint. The next thing is to restore the trochanteric group of muscles. These are restored by simply nailing the trochanter back in place with an ordinary wire nail. The next thing is to unite these muscles where I separated the fibers of the gluteus medius in front, and the anterior portion of the fibers of the gluteus maximus. You must not only have a movable joint, but a controllable one. That is why it is important to get the muscles back in accurate apposition. The bleeding is all stopped. We will not put on a ligature; we prefer not to use ligatures. That keeps the attachment all behind, so that we have good muscular apposition right from the beginning.

That completes the operation so far as the joint is concerned. The skin incision is closed with silkworm-gut and horsehair sutures. A collodion gauze dressing is applied after the wound has been dusted freely with bismuth.

The further treatment is to put the leg in an extension splint, with weight and pulley on to prevent compression and necrosis of the interposing flap. This patient is to have a twenty-five-pound weight attached to a Buck extension right away. The muscular aponeurosis was approximated with a few stitches, and that takes the place of the fascia lata, so that there is no danger of hernia on account of the muscle giving way.

The limb will be immobilized for about four weeks. We shall let him work his knee up and down, but we keep on the weight for two and a half weeks. After immobilization for four weeks we commence to put the limb through a little motion, and then we begin to lessen the traction weight when he is walking around. We put the traction on at night when he is in bed, to lessen the pressure in the joint.

Let me recapitulate the technic of the operation: First, make a U-shaped incision, with its base an inch above the tip of the trochanter major. Have the flap three inches wide and five inches long. That goes through the skin and about two-thirds of the superficial fatty tissue. An incision is then made parallel

to the long axis of the thigh from the tip of the flap downward for a distance of three inches, so as to expose a larger area of the fascia lata than is exposed by the U-shaped incision. The fascia lata is then divided through to the subfascia lata, the loose areolar tissue, clear up to the level of the base of the flap. This flap is three-quarters of an inch wider than the U-shaped flap at its lower angle, and about its width at its upper. The skin flap and the fascia lata flap are carried upward and freed from the trochanteric bursa. The fibers of the gluteus medius are then divided in front, and the neck of the femur exposed just anterior to the trochanter major; the continuation of the fascial portion of the gluteus maximus is carried upward with the flap, and the gluteus maximus fascia divided so as to expose the posterior surface of the trochanter major. The fibers are then clearly outlined, the pyriformis and obturator tendons divided as they pass down through the digital fossa anterior to the trochanter, leaving them so that they can be subsequently sutured. A chain saw is then thrown around the trochanter with all its muscular attachments intact. The trochanter is sawed off parallel to the long axis of the cervix of the femur; the capsule is then loosened from its attachment to the neck all the way round; the quadriceps femoris is maintained; the upper fibers of the lower segment of the gluteus maximus are divided; the capsule is shelled from its attachment all around the neck, but left attached to the margin of the acetabulum. With a chisel the head is freed from its attachment to the acetabulum and is molded to the normal size and shape. We divide the adductor tendon so as to get the tension off. The leg comes out into position well and will be dressed in full extension and slight abduction with the Buck extension on. The interposed fascia lata flap was sutured all around the margin of the acetabulum and to the base of the capsule, so as completely to cover the circle of the acetabulum, and is pressed into it to make a pocket into which the head of the femur is inserted. This suturing is done with phosphor-bronze wire. The femur is drawn downward, the head rotated forward, and slipped into the acetabulum and onto the interposing flap. When it is in position, the anterior and posterior portions of the

retained capsule are sutured again to the neck near the trochanter. The stumps of the obturator internus and pyriformis are reunited; the trochanter is then nailed to the femur in its normal position with a wire nail. The deep fibrous extension of the vastus externus is united with three phosphor-bronze wire sutures so as to give additional support to the margins. The separated margins of the fibers of the gluteus medius in front are then reunited, the same behind, so that the muscular connection is reëstablished on exactly the same basis that it was originally. The U-shaped skin-flap is closed with silkworm-gut and horsehair and accurately approximated. There is very little bleeding. No ligating need be done. No drain. The limb is dressed in the abducted position in a Rainey travois splint, with 25 pounds of Buck's extension applied. In putting on this travois splint you have every purpose accomplished. We have always endeavored to dress limbs in the abducted position, with the result that when we thought we had dressed the limb in an abducted position it was really an adducted position, and that is the reason for putting the limb in a Rainey splint. This splint is adjusted so that you can spread it any desired distance by loosening two bolts. It is a simple affair, and every practitioner should have it. (See Figs. 23 and 24, p. 173, vol. i, No. 2.)

The essential part in treating fractures of the neck of the femur is to keep up abduction and extension. If there is no interposition of the fibrous capsule between the fragments you will get union. If there is interposition of the fibrous capsule between the fragments there will be non-union; that is, when a fracture takes place, if the capsule is torn and, when the fragments are reduced, the capsule is pushed between the fragments, as was the condition in four consecutive cases we had here at one time, you will get no union, no matter what dressing you put on. We operated on these four cases and demonstrated our findings at the operations. We took out the capsule, put the fragments in apposition, and got perfect union in all the cases.

Let me say a few words in regard to the management of this case of Neisserian infection from its inception. First, every case of acute arthritis is a metastatic infection, and a careful

analysis in a very large percentage of the cases will enable you to locate the primary infection atrium. It does not matter whether you are able to locate it or not, if the acute arthritis involves one or two or three joints, and has associated with it a chill, the clinical diagnosis or rather prognosis is that one or two or may be more joints are to be ankylosed. Why are they going to be ankylosed? First, because of the virulence of the infection; second, from the fact that muscular contraction presses these inflamed surfaces into each other and destroys the synovial lining, and finally, the cartilage, and, third, the products of infection are held in the joint under great pressure. How do you avoid destruction of tissue from infective products in other positions in the body? If you have a phlegmon of the thigh, a phlegmon of the leg, or an osteomyelitis, what do you do? Open it, to reduce the tension caused by the products of infection. That is all you need to do. Nature cures it. The same principle applies to joints, with the exception that if you open a joint and permit the air to enter, you are going to destroy the synovial membrane. But the same principle must be carried out in relieving the tension from the products of infection in the joint and relieving the virulence by the same means, except not through a large opening, and not with the insertion of a tube.

The first thing to do when you are called to see a case of acute arthritis, whether you call it "rheumatism" or metastatic arthritis, is to relieve the intra-articular pressure and the muscular contraction, and that is done by putting on a Buck's extension. In the small joints there is but very little pressure from muscular contraction, and therefore little tendency to ankylosis. If you fracture small bones, you do not have to put on extension, as they stay in position without extension. In fractures of large bones you do put on extension. That is number one. Number two, you relieve the tension from the products of infection in the joint. This you can do by aspiration, inserting a large needle into the joint, and, without irritating or traumatizing the synovial lined surfaces, draw off the fluid. The joint cavity is a connective-tissue bottle that stands an intra-articular pressure, as demonstrated by Dr. Kellogg Speed, of from 40 to

120 pounds before it ruptures the capsule of the knee-joint and of the hip-joint. It has a non-vascular lining, which does not readily destroy the infection. That is why it holds. In other places the infection breaks through, but not at the knee or the hip, because of the leathery capsule on the outside that is non-vascular and difficult to infect.

As I have said, relieve the pressure by aspiration; render the surface and joint a bad culture-medium by injecting into the joint a fluid that stimulates polymorphonuclear leukocyte protection. An ideal remedy for that purpose is a 2 per cent. of formalin in glycerin mixture. There is a preparation of mercury, but it is too toxic to be used. Use 2 per cent. of formalin in glycerin, mixed twenty-four hours before it is injected. If you inject it into the vein of a dog, it acts as a cardiac stimulant. If you inject it into the synovial membrane, it acts, first, as a stimulator of the polymorphonuclear leukocytes, which is an important factor; second, it is a medium in which the micro-organisms will not grow, and, third, it stimulates infiltration and coffer-damming of the synovial membrane and stops absorption. One or two or three aspirations of a joint, such as we saw today, in the early stage, will avoid the necessity for opening the joint. It stops infection, and the case goes on, as we have shown here in this clinic, without the slightest tendency to ankylosis of the joint. It stops the whole thing as a primary lesion. The treatment is the same in all cases, no matter whether the primary infection is grip, pneumonia, or typhoid. They are all alike. They are all metastatic inflammations, but the clinical fact remains that the less the number of joints involved, the greater the destruction of the joints.

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